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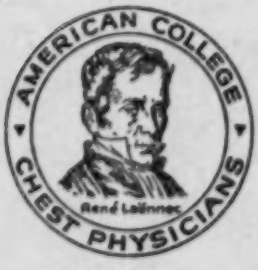
VOLUME XXVIII NUMBER 1

DISEASES

of the

CHEST

OFFICIAL PUBLICATION



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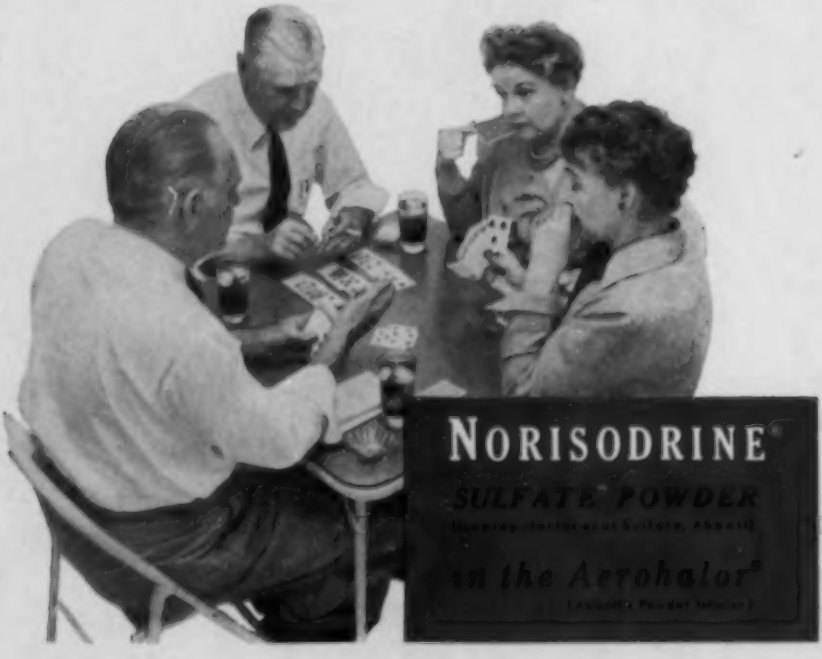

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VOLUME XXVIII JULY, 1955 NUMBER 1

CONTENTS

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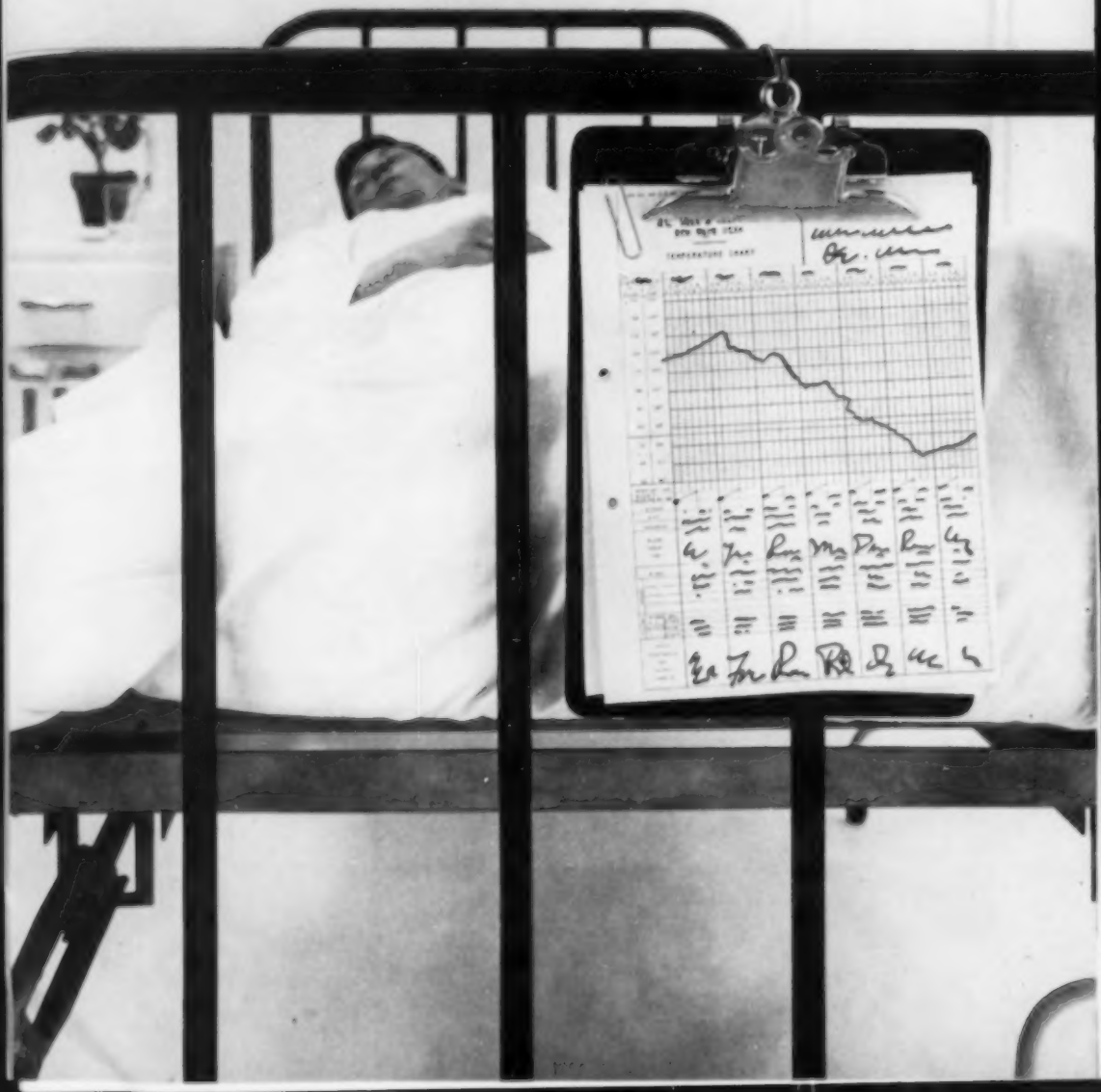

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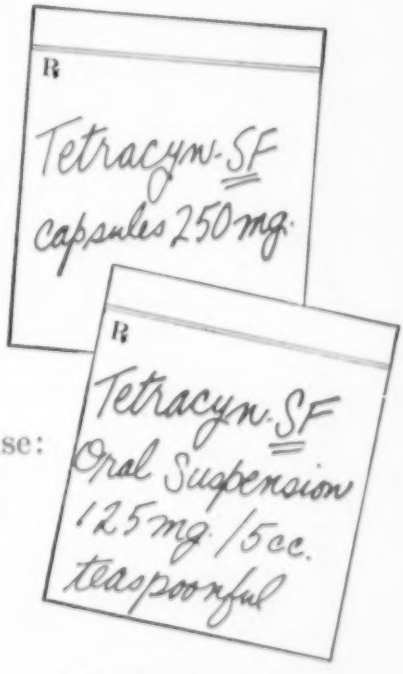

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1. Pollack, H., and Halpern, S. L.: Therapeutic Nutrition. Prepared in Collaboration with the Committee on Therapeutic Nutrition, Food and Nutrition Board, National Research Council, Washington, D. C., 1952.

2. Martí-Ibáñez, F.: Antibiotic Med. *1*:247 (May) 1955.

3. Dumas, K. J.; Carlozzi, M., and Wright, W. A.: Antibiotic Med. *1*:296 (May) 1955.

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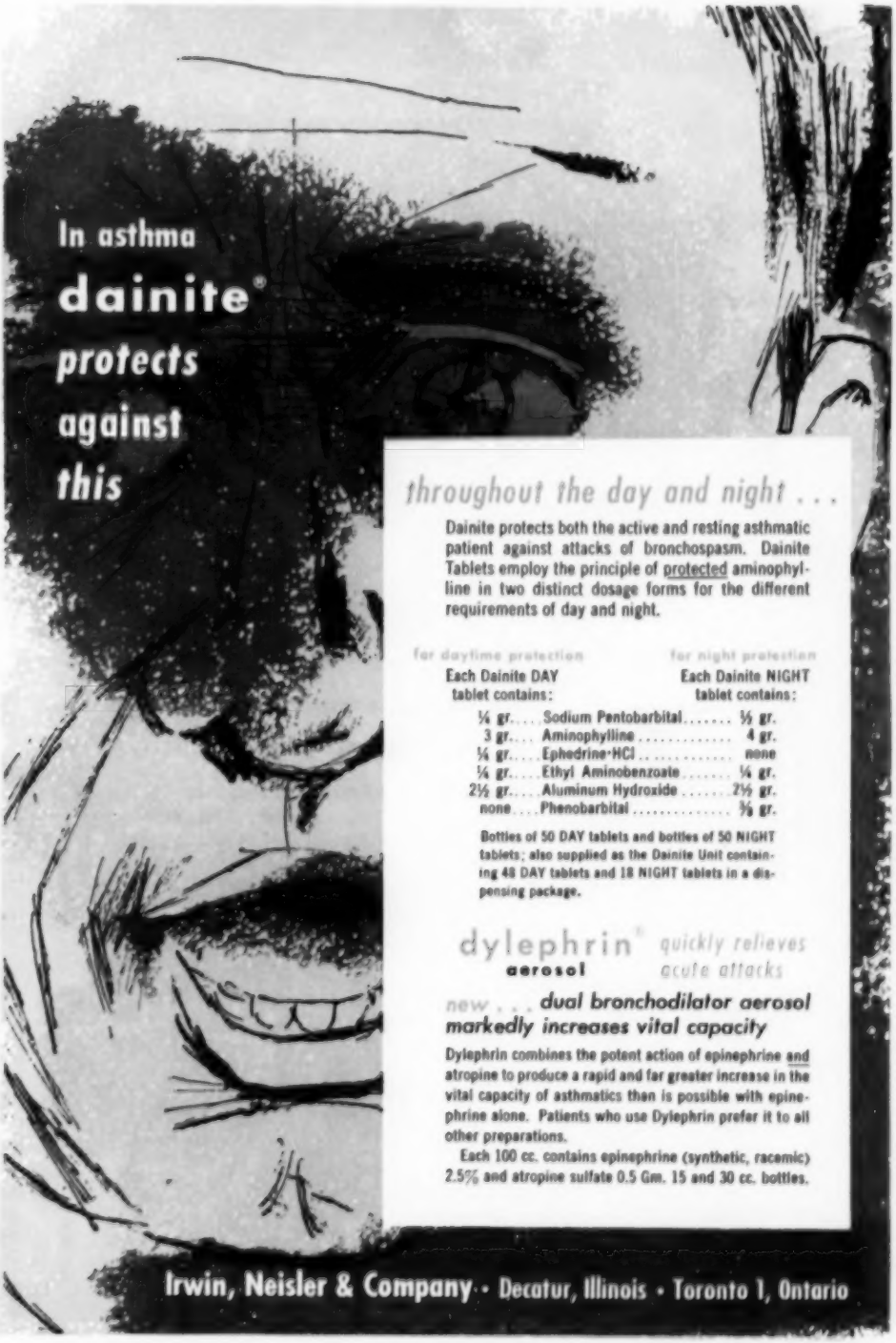

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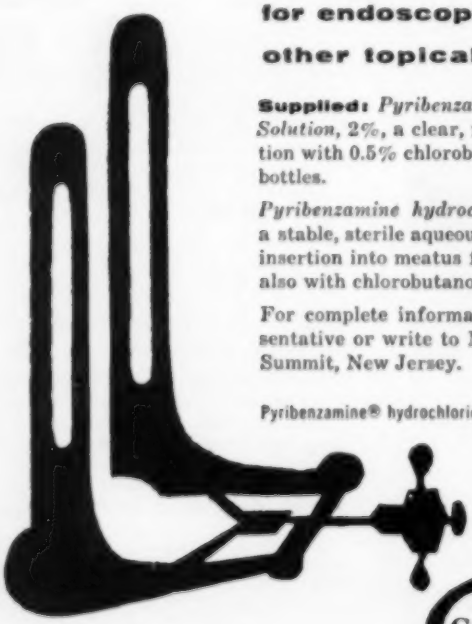

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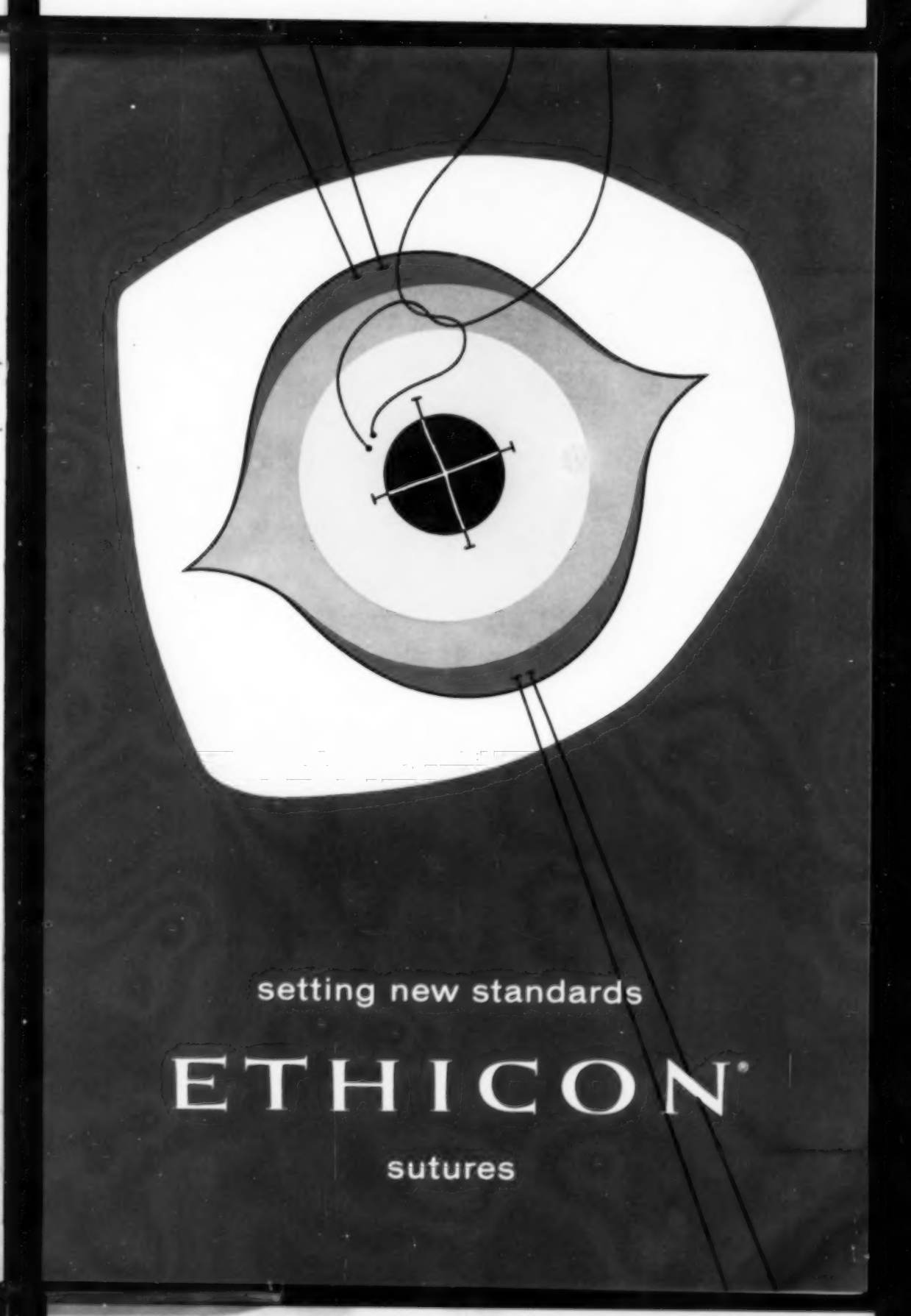
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	Mild	*Moderate*	*Total*
Streptomycin	0	0	0
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**Heck, W.E.; Lynch, W.J., and Graves, H.L.: Acta oto-laryng. 41:416, 1953.*

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DISEASES of the CHEST

VOLUME XXVIII JULY, 1955 NUMBER 1

Experiences with the Beck Operation for Coronary Artery Disease

M. W. SELMAN, M.D., F.C.C.P.

Toledo, Ohio

This article is based upon the author's observations during his participation with Dr. C. S. Beck[1] in the performance of operations for coronary disease at Mt. Sinai Hospital of Cleveland. At the time of this writing, a series of 31 consecutive patients were operated on by Dr. Beck with no mortality. Clinical evaluation of a series of 32 consecutive patients with at least six months' follow-up demonstrated the effectiveness of the operation.

The impaired coronary circulation in patients with occlusive coronary artery disease can be improved safely and effectively by the Beck operation. This improved arterial circulation in the myocardium protects the heart against death from ventricular fibrillation. The Beck operation is the result of more than 22 years of research in revascularization of the heart.[2]

Various statistical analyses attest to the high eventual mortality in patients with coronary disease.[3, 4] In the absence of any effective medical therapy, it is logical that the patient with coronary disease be given the benefit of any procedure which improves collateral circulation. The Beck operation has been demonstrated to result in an increase in intercoronary communications. It is desirable to do the operation early in the course of the disease.

To comprehend the processes by which the Beck I operation[5, 6] produces an increase in the intercoronary communications and protects the heart against ventricular fibrillation, it is necessary to understand the trigger mechanism, to become familiar with the evolution of the operation, and to know the basic experiments.

Causes of Death in Coronary Disease

Death in coronary disease ordinarily results from ventricular fibrillation or myocardial failure.

Trigger Mechanism:

Ventricular fibrillation is an arrhythmia characterized by rapid uncoordinated fibrillary twitchings of the heart muscle which makes the heart

From the Department of Cardiovascular Surgery (Service of Dr. C. S. Beck) Mount Sinai Hospital of Cleveland.

incapable of expulsion of blood. When a sudden occlusion of a coronary artery develops, the area of myocardium supplied by this artery is deprived of arterial blood and becomes ischemic. Beck[8] has labeled this area of local ischemia a "trigger" (Fig. 1). This trigger zone and the area immediately surrounding it is a highly irritable area which has the potential of discharging abnormal electrical impulses.[9] These impulses disrupt the normal coordinated contractions of the heart muscle causing ventricular fibrillation (trigger mechanism). The trigger, therefore, may be defined as a zone of local myocardial ischemia capable of causing ventricular fibrillation. Every myocardial infarct is a potential trigger area.

The production of a trigger zone and the development of the trigger mechanism can be demonstrated experimentally in the dog.[8] When the descending ramus of the left coronary artery is ligated, a zone of cyanosis develops in the area supplied by this artery. Within 15 minutes to 30 minutes, ventricular fibrillation suddenly appears and the animal dies because the heart is incapable of expelling blood. An area of ischemia as small as 10 per cent of the total heart muscle may cause ventricular fibrillation and death even though 90 per cent of the heart muscle has the potential to function normally. This is death due to a disturbance in the

FIGURE 1

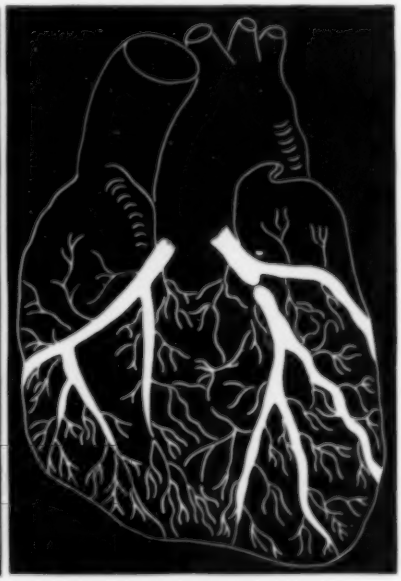

FIGURE 2

Figure 1: Drawing showing zone of local anoxia (trigger) distal to occlusion of coronary artery. Short arrows depict abnormal electrical impulses being discharged from the trigger zone. These impulses disrupt the normal coordinated contractions of the heart muscle causing ventricular fibrillation and death.—*Figure 2:* Test of Benefit Operation: Ligature around anterior descending ramus of left common coronary artery producing an acute occlusion.

normal mechanism of the heart beat and not due to a poor myocardium. Beck has named this a "mechanism death." It has been shown experimentally that introducing a small amount of arterial blood to this trigger zone will prevent the development of ventricular fibrillation. The Beck operation[3] has been shown to protect the heart against death from ventricular fibrillation.

Muscle Death:

The Myocardium, as a result of one or repeated infarcts, is gradually destroyed; and over a period of time becomes incapable of satisfactory contractions. As a result, the patient succumbs eventually to myocardial failure. Muscle death is slow as compared to the mechanism death which is sudden.

Development of the Beck Operation

In 1932, Beck[2, 3] performed experiments which demonstrated that the coronary circulation could be improved by operative methods. During the following 10 years, several thousand operations were performed on dogs.

Revascularization of the heart was accomplished in two ways. One method consisted of grafting a variety of vascular tissues on the heart so that blood vessels could grow across from the graft to the heart. Beck preferred a pedicle graft of pectoralis major muscle to the heart. Anastamotic channels could be demonstrated in some of the specimens but they were inconstant.

The second method of increasing the blood supply in the heart muscle was the production of inflammation on the surface of the heart. A bur was used to abrade the surface of the heart and pericardium. Then asbestos powder (0.2 gm.) was sprinkled over the heart. The pericardial sac was closed loosely. The abrasion and the asbestos powder caused an acute inflammatory reaction of the myocardium and pericardium. Within a few hours, the myocardium became hyperemic. This hyperemia caused dilatation of existing interarterial coronary channels and stimulated the formation of new intercoronary and extracoronary vessels. After the acute reaction subsided, a foreign body granuloma developed on the surface of the myocardium. Since the asbestos particles were not absorbed or removed by the lymphatics, they acted as a constant source of inflammation. Necropsy material in human beings and dogs showed that the granuloma was chronic, that it did not change to scar tissue, and that constrictive pericarditis did not develop.

This early Beck operation was applied to 37 humans. In 1943, Feil[10] made a clinical appraisal of the operation. There were 14 postoperative deaths. Of the 23 living patients, 83 per cent showed clinical improvement (excellent—61 per cent; good—22 per cent). Autopsies carried out on patients who died at various periods after operation showed vascular communications between the heart and surrounding tissues. In the light of our present criteria for selection of patients, many of these patients would be considered as poor candidates for coronary surgery.

Test of Benefit

A test that measured the degree of benefit consisted of ligating the anterior descending ramus of the left coronary artery at its origin about three weeks after the early Beck operation had been performed on dogs (Fig. 2). The mortality rate in this operative group of dogs was compared with that in a normal control group. Another index of benefit was the size of the infarct in the operative group compared with the normal control group. Most of the dogs died within the first hour from ventricular fibrillation. The mortality rate in the dogs that had the early Beck operation was reduced by 50 per cent and the infarcts were smaller as

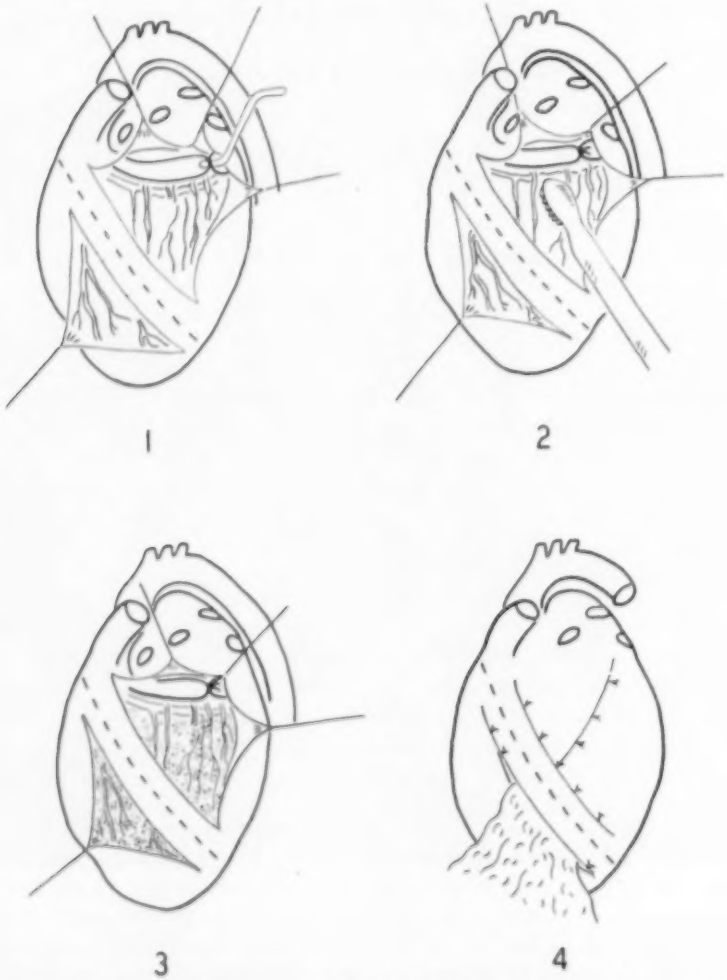

FIGURE 3: The Beck I Operation. Four steps: 1. Partial ligation of coronary sinus over 3 mm. probe; 2. Abrasion of pericardium and epicardium; 3. Asbestos powder sprinkled on surface of heart; 4. Mediastinal fat grafted to surface of heart.

shown in Table I (A): This experiment showed that this early Beck operation was beneficial and that the heart was protected.

TABLE I: BACKFLOW AND TEST OF BENEFIT

Operation	Blackflow cc./min.	No. of Dogs	Test of Benefit Mortality, Per Cent	No. of Dogs
Control Dogs	3.8 cc.	56	70	50
A. Early Beck operation:				
1. Abrasion {pericardium epicardium				
2. Asbestos powder	8.1 cc.	5	38	20
B. Beck I operation:				
1. Ligation of coronary sinus				
2. Abrasion {pericardium epicardium				
3. Asbestos powder				
4. Pedicle fat graft	8.1 cc.	43	20	10
C. Beck II operation:				
Vein graft—aorta to coronary sinus	12.0 cc.	33	8.9	45

The Beck I Operation

In 1937, Gross[11] reported the results of his work on coronary sinus occlusion. He produced complete or partial occlusion of the coronary sinus in dogs. He demonstrated that this procedure resulted in an extensive increase in the intramyocardial collateral circulatory channels. Post mortem examination showed the coronary artery channels to be wider and more abundant. This increase and dilatation in the vascular bed following coronary sinus occlusion was enough to provide significant protection from death following sudden ligation of the descending ramus of the left coronary artery in dogs. Partial ligation of the coronary sinus provided greater protection than did complete ligation.

This work was repeated by Beck,[12] who found that partial coronary sinus ligation combined with abrasion of the pericardium and epicardium and application of asbestos powder afforded greater protection than coronary sinus ligation alone. From this work, the Beck I operation evolved.

This present operation consists of four steps (Fig. 3):

1. Partial ligation of the coronary sinus.
2. Abrasion of the epicardium and pericardium.
3. Application of asbestos powder over the surface of the heart.
4. Approximation of a pedicle of mediastinal fat to the heart.

Beck applied this operation to a series of dogs. About three weeks after the Beck I operation, the Test of Benefit operation was carried out on these dogs. The mortality rate in protected dogs was reduced to 25 per cent, as compared to the mortality rate of 70 per cent in control dogs as shown in Table I (B).

The Beck Operation

In February, 1946, Beck[13] started experiments to revascularize the heart by grafting a systemic artery into the coronary sinus. This project extended over a period of two years and required an average of two operations on each of approximately 350 dogs. In its final form, the operation consisted of two stages. In the first stage, a short segment of the external jugular vein was removed from the dog's neck and grafted between the coronary sinus and the aorta. In patients, a graft of the median basilic vein was used. An orlon suture was passed around the coronary sinus near its opening into the right atrium. This suture was not tied but left loose to be tied at the second stage. Three weeks later, the second stage was performed. This consisted of opening the pleural cavity, locating the previously placed orlon suture and partially ligating the coronary sinus with this suture over a 3 mm. probe. This has been labeled the Beck II operation (Fig. 4).

This procedure produced immediate retrograde flow of arterial blood through the coronary sinus and its tributaries with retrograde perfusion of the capillary bed; thus, supplying arterial blood to areas of ischemia. The Test of Benefit operation was applied to a group of dogs about six weeks after the Beck II operation. The mortality in this group, as shown in Table I (C), was 10 per cent. This operation was applied to patients in January, 1948, about two years after the experimental study had been initiated.[14]

Physiologic and Pathologic Studies to Show the Mechanism by Which Operation Protects the Heart:

A. The Ventricular Fibrillation Experiment.—In 1952, Eckstein[15] reported the results of his experimental work showing the effect of arteriali-

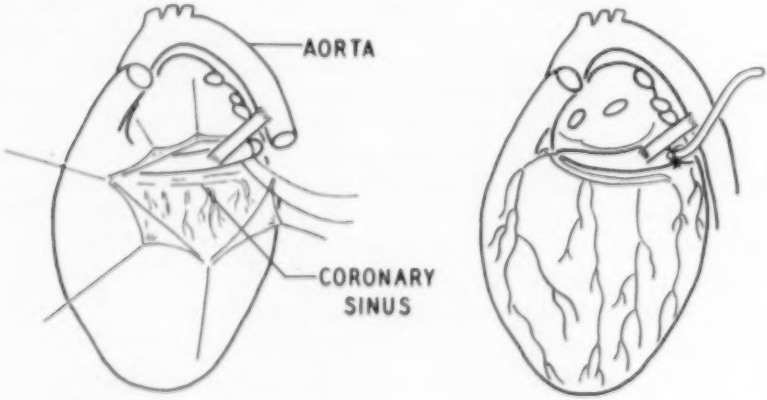

FIGURE 4: The Beck II Operation: Stage I—Approximation of vein graft between aorta and coronary sinus. Stage II—Three weeks later; partial ligation of coronary sinus over a probe.

zation of the coronary sinus on acute occlusion of the coronary artery. In a control group of dogs, the circumflex coronary artery was ligated. There was a 70 per cent mortality within one hour in this group of 20 control dogs. In another group of 20 dogs, the coronary sinus and subclavian artery were connected with a cannula. The cannula was clamped to prevent arterial blood from entering the coronary sinus. Next, the circumflex coronary artery was ligated; thus, producing an acute coronary occlusion. Then the coronary sinus was ligated, and the clamp released from the cannula. The result was a retrograde flow of arterial blood through the coronary sinus, its tributaries, and the capillary bed into the ischemic area. There were no mortalities from ventricular fibrillation in the first hour from this group of dogs (Fig. 5).

These experiments demonstrate convincingly that the introduction of a small amount of arterial blood into an ischemic area of the myocardium will prevent death from ventricular fibrillation in dogs. Since this procedure is immediately effective, it must depend upon existing vessels. The interpretation of this experiment is that any procedure that results in an increase of arterial blood to an ischemic area in the myocardium will protect the heart against death from ventricular fibrillation.[16]

B. The Backflow Experiments.—In 1953 and 1954, Eckstein[17] and Leighninger[18] reported their studies on the retrograde flow of blood through the circumflex coronary artery following arterialization of the coronary sinus. It was necessary to determine the backflow in normal dogs. The method consisted of dividing the circumflex coronary artery, ligating the proximal end and cannulating the distal end. The average backflow of the blood from the cannulated circumflex artery in a series of 41 control dogs was 3.8 cc. minute. The oxygen content of this backflow

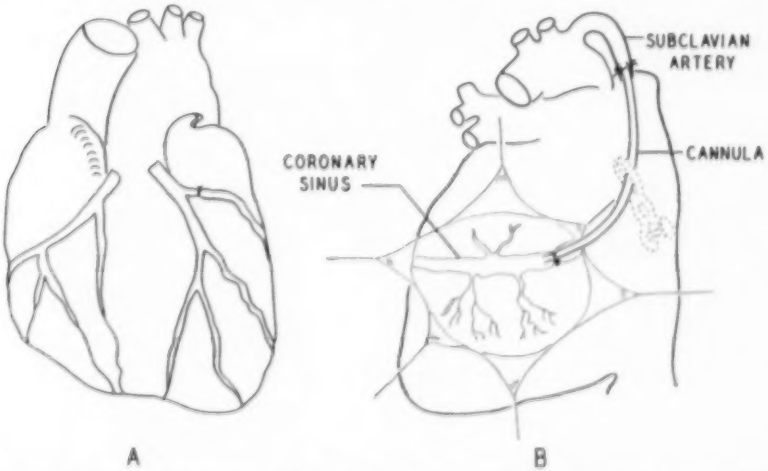

FIGURE 5: The Ventricular Fibrillation Experiment. *A*. Coronary artery ligated. Result: 70% mortality in first hour. *B*. Coronary artery ligation followed by arterialization of coronary sinus. Result: Retrograde flow of blood into ischemic area; no deaths in first hour.

blood was arterial. In another group of dogs, the Beck II operation was carried out. During the first six weeks after operation, the backflow measured 16 cc. to 28 cc. per minute. The blood was venous in nature indicating that the blood perfused through the capillary system in a retrograde manner and oxygen had been extracted. In a third group of dogs, the backflow was measured 12 to 24 weeks after the coronary sinus had been arterialized by the Beck II operation. The backflow was measured with the *graft open* and with the *graft closed* by a clamp. A significant finding was that the backflow measured 10 to 18 cc. whether the graft was open or closed. Furthermore, the blood was arterial rather than venous. This meant that the graft had lost functional contact with the capillary system and that the retrograde blood flow was due to an increase in the intercoronary communications (Fig. 6). Microscopic studies[16] revealed that the graft ultimately lost contact with the capillary bed because of obliterative changes in the tributaries of the coronary sinus. These veins revealed two principle changes: thickening of the adventitia and proliferation of the connective tissue of the intima. Backflow studies were also done on dogs which had had the early Beck operation and the Beck I operation. The findings are tabulated in Table I.

Comparison of Beck I With Beck II Operation

Since January, 1954, the Beck I operation has been performed on all eligible candidates for revascularization of the heart in preference to the Beck II procedure. There were several reasons for this change:

1. The backflow and Test of Benefit experiments showed reasonably similar results with the Beck I and Beck II operations. (See Table I.)
2. The Beck II operation is a two-stage procedure while the Back I is a one-stage procedure.

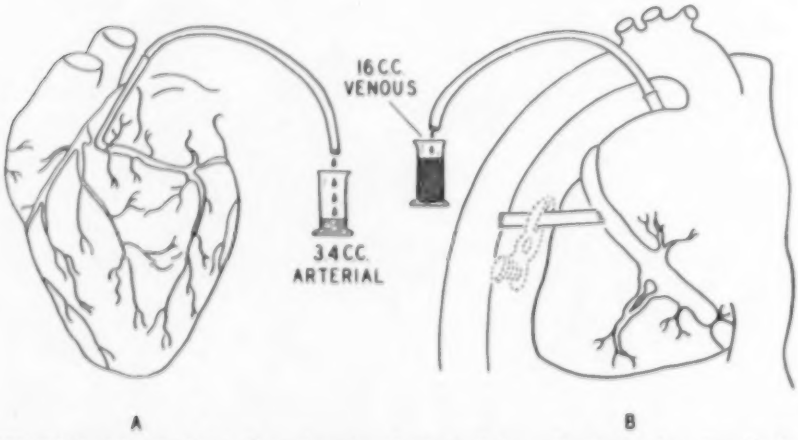

FIGURE 6: The Backflow Experiments. *A*. Control dog. *B*. Six weeks after Beck II Operation. Circumflex coronary artery ligated proximally and divided. Distal end cannulated. Amount of blood that flows back through cannula per minute is measured.

3. The Beck II is technically difficult while the Beck I is a comparatively easy operation.
4. Operating time for the Beck II totaled two to three hours; operating time for the Beck I was one and one-half hours.
5. In the dog the vein graft in the Beck II procedure lost functional contact with the capillary bed in five to six weeks. Whether this will occur in human beings is to be determined.
6. The Beck II carried a mortality of 25 per cent in man while the Beck I operation could be performed with a negligible mortality.
7. The end result of each operation appeared to be the same; namely, an *increase in the intercoronary communications.*
8. Long-term follow-up of clinical results was approximately the same in patients who had the Beck I as compared to those who had the Beck II.

Indications for the Beck I Operation

Operation is indicated where the diagnosis of coronary artery disease has been established. Usually this depends primarily on the patient's history. Physical examination, as a rule, does not contribute to the diagnosis. Electrocardiographic evidence of previous myocardial infarction helps in establishing the diagnosis of occlusive coronary artery disease. Occasionally, the electrocardiogram is normal or shows characteristic changes only after exercises. Often at operation, a myocardial scar of an old infarct is found even though the electrocardiogram showed no evidence of infarction. In a patient with a history of coronary insufficiency, operation should be considered even though there may be no objective evidence of coronary disease.

Brofman[19] has classified the selection of patients for the Beck operation into three groups:

1. Prophylactic
   Only mild angina (stable)
   Small infarct without angina
2. Therapeutic
   Moderate to severe angina (Progressive)
   Infarct followed by angina
   Repeated bouts of severe pain with or without ECG changes
3. Salvage
   Massive infarct
   Status anginosus

Most of the patients operated fall into the Therapeutic group.

Contraindication to the Beck I Operation

The Beck operation is contraindicated wherever there is extensive degeneration of heart muscle. The operation cannot repair or restore dead muscle. Therefore, nothing can be accomplished by the operation when the heart is dilated and congestive failure has developed.

The absolute contraindications may be classified as follows:

1. Recent infarct (or very severe pain) within four to six months.
2. Significant left ventricular enlargement.
3. Evidence of congestive failure.
   a. Venous pressure and circulation time elevated.
   b. Vital capacity and dynamic pulmonary function tests severely impaired.
   c. Pedal edema, dyspnea, orthopnea.
4. Marked hypertension (above 200/100).

There are certain relative contraindications to consider:

1. Borderline left ventricular enlargement.
2. Evidence of massive destruction of myocardium.
3. Possible recent infarct (even if ECG is unchanged).
4. Status anginosus, decubitus.
5. Bundle branch block on ECG.
6. Cardiac arrhythmia.
   a. auricular fibrillation.
7. Patients over 60 years of age.

Preoperative Management

Mental Attitude:

The mental attitude of the patient before coronary artery surgery deserves serious attention. Extreme anxiety may precipitate an attack manifested by angina or myocardial infarct. A few patients, while awaiting surgery, developed an acute myocardial infarct. When the anxiety is great, mild sedation with barbiturates is advisable. The patient should be reassured repeatedly about the operation. If possible, he should be allowed to talk to patients who have convalesced from the operation. An explanation of the operation in simple terms does much to allay apprehension. The author simply tells the patient that the operation is designed to create a new blood supply to the heart muscle. This explanation is adequate and apparently understood by most patients. Such talks with the patient give him confidence and expectation of success.

The Electrocardiogram:

An electrocardiogram is taken within 24 hours after admission. If surgery is to be delayed for several days, another electrocardiogram is taken on the day before the operation. If there is definite evidence of a recent infarct, surgery is deferred. A few cases showed recent infarcts after admission to the hospital while awaiting surgery. Past experience has demonstrated the prohibitive danger of operation in a patient with a recent infarct. Progress of the occlusive process may overtake the benefit from the operation. Therefore, at the present time, it is desirable to wait four to six months for stabilization of the disease.

Digitalization:

All patients are completely digitalized before surgery even though there is no congestive failure or arrhythmia. Since the employment of

preoperative digitalization, there has been a considerable reduction in alarming arrhythmias and tachycardias during the operative procedure.

The Operation

Procedure:

The patient is placed in the right lateral recumbent position. A transverse oblique incision is made from the anterior chest wall to the posterior chest wall along the line of the sixth intercostal space. The pleural cavity is entered through the sixth intercostal space. If the patient has a large barrel-type chest, the pleural cavity should be entered through the fifth intercostal space. The pericardium is opened by two parallel incisions, one on either side of the phrenic nerve, extending from the base of the heart to the diaphragm. The posterior incision is then converted into a T incision to provide access to the coronary sinus. The myocardial surface is examined for infarcts. This requires some displacement of the heart in order to visualize the diaphragmatic surface of the myocardium. Not all infarcts reach the surface of the heart. When they do, they appear as greyish white irregular areas. Next, the coronary arteries are examined be very gentle palpation and inspection. In palpating the vessels, care should be taken not to obstruct blood flow through the arteries; otherwise ventricular fibrillation may be initiated. Diseased arteries feel firm or hard. White streaks or nodules in the walls of the arteries suggest disease. The four steps on the operation are done in the following order:

1. A heavy suture of No. 1 mersilene on an atraumatic needle is passed around the coronary sinus about one centimeter from its ostium in the right auricle. The suture is not tied until Step 2 has been completed.
2. The pericardium and epicardium are abraded thoroughly with a bur designed by Beck. Then the suture is tied around the coronary sinus over a probe measuring three millimeters in diameter.
3. Sterile asbestos powder is sprinkled evenly over the surface of the heart. Not more than 0.2 Gm. is used.
4. A flap of mediastinal fat is brought in contact with the myocardium by fixing it to the under surface of the pericardium; and the pericardium is closed loosely with interrupted silk.

After the coronary sinus has been partially ligated, the normal red of the myocardium becomes dark due to partial obstruction of the venous return, and the superficial veins appear distended. Persistent bleeding from the surface of the heart muscle can be controlled by suturing a free graft of muscle tissue over the bleeding area. The suture is snug enough to control the oozing but not so tight as to obstruct coronary blood flow.

A large rubber catheter is inserted into the pleural cavity through a stab wound in the eighth intercostal space in the posterior axillary line. The catheter is connected to a water trap system. The chest wall is closed in layers.

Operative Management

Anesthesia:

Anesthesia for the cardiac patient is the same as anesthesia for any intrathoracic procedure. To achieve smooth induction, pentothal is preferred. Other anesthetic agents such as ether, nitrous oxide, or cyclopropane are used depending on the experience and preference of the anesthetist. Adequate ventilation is needed to afford maximal oxygenation of the myocardium and to avoid hypercapnia which predisposes to ventricular fibrillation. The Rand-Wolfe respirator has provided excellent ventilation of the lungs in the series of cases reported in this paper.

Hypotension:

The blood pressure during the operation is checked frequently. Hypotension in the presence of coronary artery disease may result in thrombosis in the partially occluded vessels. Blood loss during this procedure is so small that it cannot be considered a cause for hypotension.

Peripheral vasodilatation that occurs in general anesthesia may cause severe hypotension. Myocardial impairment due to the damaged heart muscle may cause hypotension. It is important to treat hypotension under anesthesia as soon as it develops. The first step is to stop the operation and manipulation of the heart; the lung is re-expanded and hyperventilated. If the hypotension is not corrected in a few minutes, neosynephrine, vasoxyl Rx or wyamine Rx should be given intravenously. Thus far, there has been no patient in this series in whom hypotension was intractable.

Blood and Fluid Replacement:

Replacement of blood loss has been no problem. The average patient loses about 250 cc. to 300 cc. of blood during the operation. For this reason measurement of blood loss by the sponge-weight method has not been used. Overtransfusion is probably more injurious to the damaged myocardium than is a small deficit of a few hundred cubic centimeters of blood.

Infusions of glucose or saline solution over 500 cc. are avoided during the operation. A small deficit in fluids is preferable to overloading the circulation in the presence of a damaged myocardium.

Electrocardiographic Behavior of the Heart During Operation:

The patients are connected to a cardioscope before operation is started so that every heart beat can be monitored. Practically all the patients show some slight disorder of the heart beat. Many of the arrhythmias that occur are common to thoracic surgery in general; and, no doubt, related to anesthesia, reflexes, hypoxia, and the incision.

Bradycardia frequently occurs while the chest is being opened. This is probably due to vagal stimulation. The heart rate during induction of anesthesia may drop below 60 per minute in some cases. In most cases, the bradycardia can be corrected with intravenous atropine sulfate. Usually, a progressive increase of the heart rate is noted. A sinus tachycardia above 110 occurs in many cases shortly after the pericardium is opened.

Usually the heart rate decreases if the operation is stopped and the lung inflated. Such rest periods are important in the prevention of serious tachycardias and arrhythmias. Besides rest periods, inflation and hyperventilation of the lungs, cedilanid or prostigmine can be given intravenously to control the tachycardia.

Disturbance of the cardiac mechanism during induction of anesthesia is characterized by displacement of the pacemaker, nodal rhythm, or interference dissociation. Premature auricular and ventricular beats occur frequently prior to and during the opening of the chest. These premature contractions generally occur as single beats. Runs of premature beats occasionally develop during manipulation of the heart. Although anesthetic agents alone may be responsible for premature beats, transient hypoxia is probably the most important cause of these contractions. These arrhythmias are usually transient.

Ventricular Fibrillation:[20]

This is almost always a terminal event. However, every effort should be made to defibrillate the heart. To reduce the irritability of the heart, 10 cc. of a 1 per cent solution of procaine should be injected into the right ventricular cavity, the heart should be massaged then shocked into standstill with a defibrillator. This should be followed with vigorous intermittent manual compression. Massage and electrical defibrillation should be repeated as necessary.

Cardiac Standstill:[20]

Should this occur, cardiac massage is instituted and the lung is ventilated with 100 per cent oxygen. This procedure usually restores the heart beat. If persistent, one cc. of 1:1000 adrenalin solution diluted with 10 cc. of saline is injected into the ventricular chamber.

Once cardiac arrest occurs in a patient with severe coronary disease, attempts at resuscitation are rarely, if ever, successful.

General Measures:

The general measures applied to all patients subjected to thoracic surgery are employed in the postoperative care following the Beck operation. The changed physiology of the open chest is corrected by an air-tight closure of the chest wall, inflation of the lung, and the evacuation of fluid and air from the pleural cavity by means of a drainage tube connected to a water-trap system. Penicillin and streptomycin are given the day before surgery and continued for several days following the operation.

Oxygen:

Oxygen, six to eight liters per minute via a nasal catheter inserted through a small foam rubber plug, is administered for at least the first 24 hours. However, the oxygen is resumed if the pulse or respiratory rate become elevated. It may be necessary to continue the administration of oxygen for four or five days. Because of the dangerous effect of anoxia on the myocardium, adequate ventilation of the lungs is important.

Air Passages:

The air passages should be kept clear of tracheobronchial secretions by encouraging the patient to cough frequently while in the sitting position.[17] If this does not clear the rales and rhonchi, nasotracheal suction should be employed. Demerol is used in sufficient dosage to allow the patient to cough comfortably and breathe deeply.

Hypotension:

Some patients develop hypotension immediately after surgery. If the pressure goes below 100 systolic, wyamine Rx is administered periodically to maintain an adequate pressure.

Pleural Complications:

Pleural effusion occurs frequently. Occasional thoracentesis may be required, but usually spontaneous absorption occurs.

Physiotherapy:

Ambulation is started about the third postoperative day. Physiotherapy is started four days after surgery so that the patient has free motion in the shoulder girdle. Persistent pain in the shoulder girdle is frequently due to fixation and muscle spasm. It may be difficult to differentiate this pain from angina. Properly supervised physiotherapy will minimize postoperative shoulder girdle pain.

Pleuropericardial Pain:

Within two to three weeks following surgery, some patients develop a severe chest pain that simulates the pain of myocardial infarcation or acute pericarditis. The pain is precordial and radiates to the back, shoulders, and down either arm. Change of position and deep breathing aggravate the pain. It is a persistent type of pain that requires narcotics for relief. The pain usually clears within two to three days. However, it may recur during the next several months. The electrocardiogram shows no changes over the previous record.

Postoperative Period:

Most patients are discharged by the 14th postoperative day.

Results

Operative Mortality:

At the time of this writing, 31 consecutive patients have been operated without operative or immediate postoperative mortality. In a larger series, Brofman[19] found a mortality rate of approximately 5 per cent for the Beck I operation.

Clinical Evaluation:

A clinical evaluation was carried out on 32 consecutive patients who had gone six months or more since operation. Evaluation was based primarily upon such factors as relief from pain, sense of well-being and ability to work. The duration of symptoms had ranged from four months to 13 years. All but six of the 32 patients had at least one clinically proved

myocardial infarction, and seven had two or more infarctions. All but one had angina.

Brofman summarized the postoperative cardiac status of these 32 patients as follows:

1. Excellent	10 (31.3)	
2. Good	12 (37.5)	87.5 Per Cent
3. Fair	6 (18.7)	Improved
4. Unimproved	4 (12.5)	

He defined the classification as follows:

1. Excellent: able to perform full-time work with little or no pain.
2. Good: able to perform full or part-time work with significant reduction in pain.
3. Fair: able to perform more work than before operation with some dimunition of pain.
4. Unimproved: unable to work up to the present. Evaluation in this group has been complicated by such factors as narcotic addiction, congestive failure, and psychosis.

Since many of the patients have shown progressive improvement, the ultimate evaluation must be made after more time has elapsed. At the present time, clinical assessment has depended upon subjective evidence such as the patient's relief from pain, his ability to work, and his sense of well-being. Recently Brofman has been doing ballistocardiograms on patients before and after the Beck operation. This study may prove to be a valuable objective test.

For purposes of illustration, a typical case history follows:

A 45-year-old white female was admitted to the hospital complaining of precordial pain. She had two myocardial infarctions in the year prior to operation. During this period, she had precordial pain almost continuously. The pain varied in intensity from mild to severe; and lasted from a few seconds to several minutes. Nitroglycerine frequently relieved the pain. Cold weather aggravated the pain. Walking less than a block or climbing a flight of stairs produced the pain. Emotional disturbances precipitated an attack of pain. Frequently she experienced pain while at rest. She experienced no nocturnal dyspnea and no dyspnea lying flat in bed. However, walking a short distance or climbing stairs caused mild dyspnea. There was no cough, no hemoptysis, no wheezing, and no pedal edema. The patient was a hotel proprietress; almost totally disabled in her occupation because of the pain.

Examination revealed a well developed, well nourished white female. There was no apparent dyspnea, no cyanosis, no clubbing of the fingers and no pedal edema. Examination of the heart revealed the apex impulse and left border of cardiac dullness to be within normal limits. There was regular sinus rhythm with a rate of 70 per minute. Heart tones were of good quality. There were no murmurs. Blood pressure was 110/70. Venous pressure was 90 mm of water. Arm to tongue circulation time (Decholin) was 20 seconds. Blood count and urinalysis were normal.

x-ray film of chest and cardiac fluoroscopy showed no definite cardiac enlargement. The electrocardiogram showed old posterolateral and anteroseptal myocardial infarcts.

On April 28, 1954, a Beck I operation was performed. An old infarct was visible at the apex of the heart. The left common coronary artery felt soft; however, the anterior descending branch felt firm and indurated. The patient's postoperative course was satisfactory and uneventful. She was discharged 13 days following surgery. She showed progressive improvement in her symptoms. Eight months following surgery she was free of pain and able to return to her former occupation.

Discussion

Many patients with coronary artery disease die of ventricular fibrillation. Yater[21] found that one-third of all victims of coronary artery disease showed no myocardial infarcts either old or recent. Apparently, these patients died a mechanism death. There is another group of patients with small infarcts who develop mechanism deaths. In each of these groups of patients, the myocardium had the potential for continued function, yet, the patients died because of a disruption in the normal mechanism of the heart beat. On the basis of laboratory experiments, it is believed that the Beck I operation will protect the heart against death from ventricular fibrillation.

Another cause of death in coronary artery disease is the muscle death. As a result of one or more myocardial infarcts, the heart muscle is replaced by scar tissue. Eventually, enough myocardium is destroyed so that the heart is unable to function properly and the patient dies of cardiac failure. The experiments on dogs carried out by Beck and associates demonstrated a decrease in the number and size of myocardial infarcts following acute occlusion of a major coronary artery in dogs which had the Beck I operation. From these experiments, it is reasoned that the number and size of myocardial infarcts following a coronary occlusion will be less in those patients previously protected by the Beck I operation. This, of course, means a delay in the muscle death and, therefore, a longer life for the patient.

Zoll, Wessler, and Schlesinger[22] studied 1,050 human hearts at necropsy and came to the conclusion that functionally, the coronary arteries are end arteries. They found intercoronary arterial anastomoses in nine per cent of the normal hearts, in 58 per cent of the hearts with recent coronary occlusions, and in 96 per cent of the hearts with old coronary occlusions. There was a striking increase in the intercoronary anastomoses in occlusive coronary artery disease. Apparently, coronary occlusion stimulates the coronary arterial system to develop functioning internal anastomotic collateral circulation to compensate for a relative insufficiency of blood. The incidence of coronary artery anastomoses following acute coronary occlusion was much less than the interarterial anastomoses in old coronary occlusion. In the recent coronary occlusions the intercoronaries probably developed less rapidly than did the occlusive process.

apparently, the greater the time interval after a coronary occlusion, the greater is the incidence of compensatory anastomoses. These authors found a few hearts with complete coronary artery occlusions but without any myocardial infarcts. This finding illustrates the protective value of intercoronary anastomoses in preventing organic muscle damage distal to complete occlusion of a coronary artery.

Although the myocardium produces new collateral coronary anastomoses following coronary artery occlusion, the rate of development of these new collaterals is inadequate in most cases to keep up with the rate of occlusion produced by the disease process. Therefore, any treatment which can augment and speed up the development of intercoronary communications is desirable. By causing hyperemia and the development of a foreign body granuloma around the heart, the Beck operation stimulates the rapid development of intercoronary anastomoses. Just how the occlusion of the coronary sinus in this operation causes an increase in the interarterial collateral channels is not clear. There appear to be many factors in the Beck I procedure which stimulate an increase in intercoronary communications. These factors are being investigated in the experimental laboratory of Dr. Beck at Western Reserve University.

Physiological and anatomic studies of the Beck operations[15, 16, 17, 18, 23, 24, 25] have shown the following mechanisms by which the blood supply to the myocardium is increased:

1. The approximation of vascular tissues to the myocardium provides a variable degree of extracoronary arterial blood to the myocardium.
2. Abrasion of the pericardium and epicardium and the application of asbestos powder to the myocardium results in the development of a chronic granuloma and an increase in the intercoronary and extracoronary communications.
3. Ligation of the coronary sinus stimulates a rapid increase in interarterial coronary anastomoses.
4. The anastomoses of a blood vessel graft between the aorta and coronary sinus results in a temporary retrograde flow of arterial blood through the myocardium; and eventually results in an apparently permanent increase in the interarterial coronary channels.

Other operations described in the literature[26, 27, 28, 29, 30, 31] to increase the blood supply to the myocardium are variants of the Beck operation and produce beneficial results by one or a combination of these mechanisms.

SUMMARY

1. There are two common causes of death from occlusive coronary artery disease:
   a. Ventricular fibrillation (Trigger Mechanism), which is sudden.
   b. Myocardial failure (Muscle Death), which is slow.
2. There is ample experimental evidence that *the Beck I operation will protect the heart against death from ventricular fibrillation* and will prevent ischemic areas responsible for angina.

3. The number and size of myocardial infarcts after coronary occlusion were significantly reduced in dogs protected by the Beck I operation.
4. The Beck operation cannot stop the degenerative and occlusive disease in the coronary arteries.
5. The Beck procedure cannot restore degenerated myocardium and is therefore contraindicated in cases where destruction of the myocardium from infarcts has resulted in myocardial failure.
6. The occlusive coronary artery disease may be so rapidly progressive that it may overtake the benefit derived from the operation.
7. The Beck operation *results in an increase in the intercoronary and extracoronary communications* thereby augmenting the natural tendency of the heart to increase blood supply to the myocardium following coronary occlusion or narrowing.
8. In approximately 80 per cent of the patients who were evaluated following the Beck operation, the result has been excellent to good.
9. There were no operative or immediate postoperative mortalities in a series of 31 consecutive patients operated.
10. There is ample experimental and clinical evidence to warrant the widespread application of the Beck operation to properly selected patients with coronary artery disease.

RESUMEN

1. Hay dos causas comunes de muerte por la enfermedad coronaria oclusiva: a. Fibrilación ventricular (mecanismo de gatillo) que es repentina. b. Insuficiencia del miocardio (muerte muscular) que es lenta.

2. Hay amplia evidencia experimental de que *la operación de Beck I proteje el corazón contra la muerte por fibrilación ventricular* y prevendrá las áreas de isquemia responsables de angina.

3. El número y el tamaño de los infartos del miocardio después de oclusión coronaria se reduce significantemente en los perros protejidos por la operación Beck I.

4. La operación de Beck no puede detener la enfermedad degenerativa y oclusiva en las coronarias.

5. El procedimiento de Beck no puede regenerar el miocardio degenerado y está por tanto contraindicada en los casos en que la destrucción del miocardio por infartos ha resultado en insuficiencia del miocardio.

6. La enfermedad coronaria oclusiva puede ser tan rápidamente progresiva que puede sobrepasar el beneficio derivado de la operación.

7. La operación de Beck *produce un aumento de las comunicaciones intercoronarias y extracoronarias*, aumentando por tanto la tendencia natural del corazón a aumentar la irrigación sanguínea al miocardio después de oclusión coronaria o estrechamiento.

8. El resultado después de la operación de Beck ha sido desde excelente a bueno en el 80 por ciento de los enfermos.

9. No hubo mortalidad operatoria o postoperatoria inmediata en una serie de 31 enfermos operados consecutivamente.

10. Hay evidencia experimental y clínica que respalda el uso amplio de la operación de Beck en los casos bien seleccionados de enfermedad arterial coronaria.

RESUME

1. La thrombose des artères coronaires peut entrainer la mort de deux façons habituelles: 1) par fibrillation ventriculaire et dans ce cas, la mort est subite; 2) par insuffisance myocardique et dans ce cas, l'évolution est lente.

2. L'expérimentation a mis en évidence d'une façon certaine que l'opération de Beck peut prévenir la fibrillation ventriculaire mortelle, et peut empêcher la constitution de zones ischémiques qui déterminent l'angor.

3. La quantité et les dimensions des infarctus myocardiques produits chez le chien après occlusion coronarienne, furent considérablement réduites grâce à l'opération de Beck.

4. L'opération de Beck n'est pas susceptible cependant de mettre obstacle à la maladie dégénérative et thrombosante des artères coronaires.

5. L'opération de Beck ne peut pas réparer le myocarde dégénéré. C'est pourquoi elle est contrindiquée lorsque l'atteinte myocardique due à l'infarctus a entrainé une insuffisance du muscle.

6. Il est possible que la maladie thrombosante des artères coronaires puisse évoluer si rapidement qu'elle ne permette pas à l'intervention d'amener une résolution.

7. L'opération de Beck consiste à augmenter les anastomoses inter et extra-coronariennes en utilisant la tendance naturelle du coeur à augmenter l'apport sanguin vers le myocarde après occlusion coronarienne.

8. Pour environ 80% des malades, l'opération de Beck donna des résultats excellents ou bons.

9. Il n'y eut ni mortalité opératoire ni mortalité post-opératoire immédiate, dans une série de 31 malades.

10. Les résultats expérimentaux et cliniques permettent de répandre l'opération de Beck chez les malades atteints d'affection coronarienne, à condition qu'ils soient judicieusement choisis.

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A Tuberculin Survey in a Metropolitan District and its Relation to Morbidity and Mortality*

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Introduction

Decrease in the death rate from tuberculosis is only the first step in the process of eliminating this disease from a community. The second step is the reduction in the number of new cases, and the final and decisive step is the reduction in the number infected with tuberculosis, as shown by a falling tuberculin-reactor rate. In order to know the tuberculosis situation in this community, a tuberculin survey was made and the new clinical cases of disease and death occurring during the same period were analyzed. In this way, any relationship between the tuberculin survey and the number of new cases and deaths was determined.

Method and Material

During the calendar years, 1949 and 1950, all new admissions to the Jamaica Chest Clinic of the New York City Health Department received a Mantoux test of 0.1 cc. of 1:1000 Old Tuberculin. The test was read in 72 hours, and an induration of 5 mm. or more was considered as positive. Those who came in with a known diagnosis of tuberculosis were excluded from the survey. New cases and deaths occurring during the same years, 1949 and 1950, in the Jamaica Health District, which is coterminus with the Jamaica Health Clinic, were tabulated.

The Jamaica Chest Clinic admits a variety of patients: contacts to tuberculosis cases, those sent in by private doctors for consultation or routine x-ray film inspection, and also those who come by themselves for a routine x-ray film check-up with or without symptoms. The Jamaica district, one of the districts of New York City, had a total population in the 1950 U. S. census of 533,702, 6.4 per cent of whom were non-white.

Results

Tuberculin Survey

Table I shows the number and per cent of reactors by age, color and sex. Fig. 1, compiled from the same data, gives a graphic picture. It is noted that infection in the first 15 years of life is not negligible; that the rates for males and females are similar; and that the rates for non-whites are higher than those for whites. There is a falling off in the positive reaction in the group 65 years of age and over. This decline in the older age group has been noted before[1] and suggests a probable loss in skin sensitivity.

*From the New York City Health Department, Bureau of Tuberculosis.

TABLE I

TUBERCULIN TESTS IN JAMAICA DISTRICT CLINIC, 1949-1950
NUMBER & PERCENT POSITIVE BY AGE, SEX, AND COLOR

— A —

Age	Total					
	Male			Female		
		Positive			Positive	
	Number Tested	No.	Per Cent	Number Tested	No.	Per Cent
0- 4	234	31	13.2	228	23	10.1
4- 9	257	40	15.6	268	44	16.4
10-14	158	46	29.1	194	58	29.9
15-19	136	40	29.4	266	68	25.6
20-24	148	62	41.9	446	198	44.4
25-29	173	102	59.0	453	222	49.0
30-34	158	111	70.3	380	218	57.4
35-44	243	176	72.4	480	341	71.0
45-54	178	146	82.0	280	224	80.0
55-64	116	96	82.8	167	132	79.0
65	63	46	73.0	73	48	65.8
Unk.	54	6	11.1	56	9	16.1
	1918	902	47.0	3291	1585	48.2

— B —

Age	White						Non-white					
	Male			Female			Male			Female		
		Positive			Positive			Positive			Positive	
	Number Tested	No.	Per Cent	Number Tested	No.	Per Cent	Number Tested	No.	Per Cent	Number Tested	No.	Per Cent
0- 4	182	22	12.1	166	18	10.8	51	9	17.6	61	5	8.2
4- 9	204	28	13.7	210	31	14.8	53	12	22.6	57	13	22.8
10-14	126	33	26.2	153	43	28.1	30	11	36.7	38	13	34.2
15-19	107	29	27.1	167	33	19.8	28	10	35.7	98	35	35.7
20-24	113	44	38.9	266	100	37.6	35	18	51.4	179	98	54.7
25-29	127	67	52.8	277	119	43.0	44	33	75.0	173	101	58.4
30-34	126	87	69.0	278	150	54.0	30	22	73.3	102	68	66.7
35-44	201	144	71.6	368	252	68.5	40	30	75.0	110	88	80.0
45-54	154	124	80.5	231	184	79.7	23	22	95.7	47	39	83.0
55-64	104	85	81.7	139	110	79.1	12	11	91.7	28	22	78.6
65	54	38	70.4	66	42	63.6	9	8	88.9	7	6	85.7
Unk.	38	6	15.8	40	7	17.5	15	—	0	16	2	12.5
	1536	707	46.0	2361	1089	46.1	370	186	50.3	916	490	53.5

Differences in Positive Tuberculin Reaction Rates of Contacts and Non-Contacts

Table II groups these findings in the tuberculin survey into larger aggregates and shows the number and per cent positive in contacts and non-contacts. Contacts were limited as far as possible to those in contact with an active case of tuberculosis, but this information is not entirely reliable and also included those in which the state of the source was not determined. Fig. 2 is a graphic presentation of this data. It is apparent that from 30 years on, there is no marked difference between contacts and non-contacts. Between the periods of 0 and 14 years, the rate is definitely higher for contacts than that of the remaining groups. This suggests that by adult life most of the non-contacts have been infected

TABLE II
TUBERCULIN RATES FOR CONTACTS AND NON-CONTACTS BY AGE AND SEX

Age Groups In Years	Sex	Contacts			Non-contacts		
		Number Tested	Positive		Number Tested	Positive	
			Number	Per Cent		Number	Per Cent
0 to 14	M	210	54	26.2	425	59	13.6
	F	206	59	28.6	471	69	14.7
15 to 29	M	94	39	41.5	345	155	44.9
	F	134	73	61.9	1003	404	40.3
30 to 44	M	70	49	70.0	318	229	72.0
	F	141	100	70.9	706	451	63.9
45 to 64	M	45	39	82.2	234	191	81.6
	F	124	98	79.0	308	245	79.5
65	M	11	8	72.7	50	36	72.0
	F	22	14	63.6	51	34	66.6

in this community by meeting undiagnosed cases of tuberculosis in the general population.

Analysis of Cases and Deaths in the Jamaica District In 1949-1950 and the Relation to Tuberculin Reaction Rates

Table III gives the numbers and rates of clinical cases and deaths from tuberculosis in the Jamaica District, 1949-1950, and also in the population assumed to have a positive tuberculin reaction; assuming that the rates found in our clinic survey represent the general population. This assumption will be discussed later.

Figs. 3 to 7 show the same data in graphic form. As noted from Fig. 3, it is apparent that in this community there is little difference by sex in the positive tuberculin reaction rate using larger aggregates, and that

there is a steady increase in the rates to a maximum in the 45 to 64 year groups and a falling off in the rates for the groups 65 years and over, the latter being probably due to a loss of skin sensitivity.

Fig. 4 shows that the case rates are low in the age group up to 14 with females outnumbering males. In the group, 15 to 29, the rate rises abruptly. It is in this age group that the maximum rate of 78.8 new cases per 100,000 population occurs in females; after which it falls for females to 33.2 in the age group 45 to 64. The male case rate rises steadily to a maximum of 133.8 in the group of 65 and over. In the age group 45 to 64, the male rate is over three times the female rate. As mentioned before, non-whites comprise only 6.4 per cent of the population in this district. However, they comprise 22.5 per cent of all newly diagnosed cases during this study. It is also worth knowing that of all the known registered

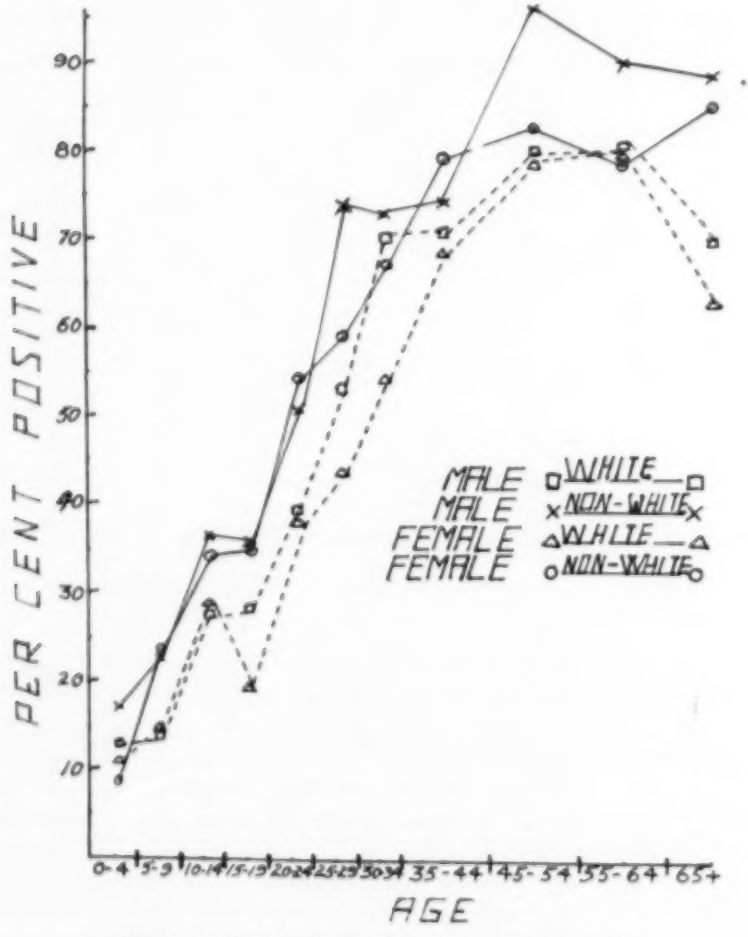

FIGURE 1: Tuberculin reactors by age, sex and color.

TABLE III
NUMBER OF CLINICAL CASES OF AND DEATHS FROM TUBERCULOSIS OCCURRING IN THE TOTAL POPULATION AND ALSO ASSUMED INFECTED POPULATION OF JAMAICA—1949-1950

— A —

Age Groups In Years	Sex & Total	Total Population Jamaica 1950	Per Cent Positive In Tuberculin Tests	Population Assumed to Have Positive Tuberculin Reaction
0	M	59,438	18.0	10,699
to	F	57,083	18.1	10,332
14	Total	116,521		21,031
15	M	55,593	44.6	24,794
to	F	60,277	41.9	25,256
29	Total	115,870		50,050
30	M	58,246	71.6	41,704
to	F	65,438	65.0	42,535
44	Total	123,684		84,239
45	M	67,154	82.3	55,268
to	F	69,181	79.6	55,068
64	Total	136,335		110,336
65	M	18,320	44.4	8,134
	F	22,972	44.2	10,154
	Total	41,292		18,288
Grand Total		533,703		283,944

— B —

		Cases			Deaths		
Age Groups In Years	Sex & Total	Number Reported 1949-50	Case Rate Per 10,000 Infected Population	Per 100,000 Population	Number Reported 1949-50	Death Rate Per 10,000 Infected Population	Per 100,000
0	M	14	6.6	11.8	3	1.4	2.5
to	F	22	10.7	19.1	5	2.2	4.4
14	Total	36	8.5	15.4	8	1.9	3.4
15	M	77	15.5	69.3	9	1.8	8.1
to	F	95	18.4	78.8	14	2.8	11.4
29	Total	172	17.1	74.2	23	2.3	10.0
30	M	82	9.8	70.4	33	3.7	28.4
to	F	93	10.9	71.1	25	2.9	19.1
44	Total	175	10.4	70.7	58	3.4	23.5
45	M	150	13.6	111.6	83	7.5	61.8
to	F	44	4.0	33.2	19	1.7	13.7
64	Total	194	8.8	71.1	102	4.6	37.4
65	M	49	30.1	133.8	25	15.4	68.3
	F	21	10.3	45.6	16	7.9	34.8
	Total	70	19.1	84.7	41	11.2	49.6
Grand Total		647			232		

cases of tuberculosis in this area, living as of December 31, 1949, 13.2 per cent of the known cases registered were non-white.

In the total assumed infected population (Fig. 5) the peak of new cases is again 15 to 29 age group in the females. This peak is more marked than seen in Fig. 4 in the general population and emphasizes the danger of developing clinical disease in the adolescent infected female. The marked increasing number of new cases in the males over 45 is also noted. The peak is probably exaggerated in the group 65 and over, because of the fact that the tuberculin tests in this group of 65 and over does not actually measure the number previously infected, since many probably have lost their allergy. Nevertheless, a positive tuberculin test is significant, since if found at this age the higher death rate would apply.

A study of the death rates (Fig. 6) shows that the rates are low from 0 to 14. They rise steadily, and the peak rate 19.1 per 100,000 among females is reached at 30 to 44 and then falls in the next age group. Among the males, the death rate rises steadily from 30 years onward and is higher than for females. From 45 to 64 the death rate 61.8 among males is more than four times that for females. It is worth pointing out that of all the deaths in this community during this study, 17.7 per cent occurred

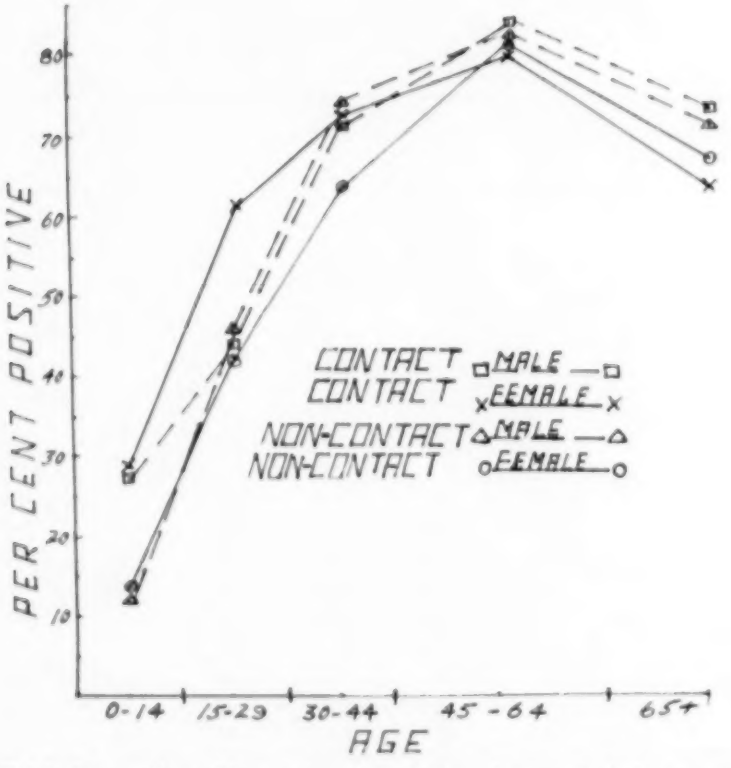

FIGURE 2: Tuberculin positive reactors in contacts and non-contacts by age and sex.

among non-whites, whereas non-whites contribute only 6.4 per cent of the total population.

An analysis of the death rate of the infected population (Fig. 7) points up again the relatively low rate (from tuberculosis) in childhood and adolescence. Early in life, females outnumber males, but from 45 to 64 the male rate is over four times that of the female rate. The rates at age 65 and over are again exaggerated due to the abnormally low tuberculin positive rates in this group. However, it emphasizes that finding a positive test in an elderly person is significant with an increasing probability of developing disease and dying from tuberculosis.

Comment

Statistically it must be admitted that a tuberculin survey based on clinic population is not truly representative of the findings in the general population. A true random selection of population groups according to age, race and sex as distributed in the general population is impractical in this community. In order to determine which portion of an age group that is positive had been previously infected in an earlier group, would

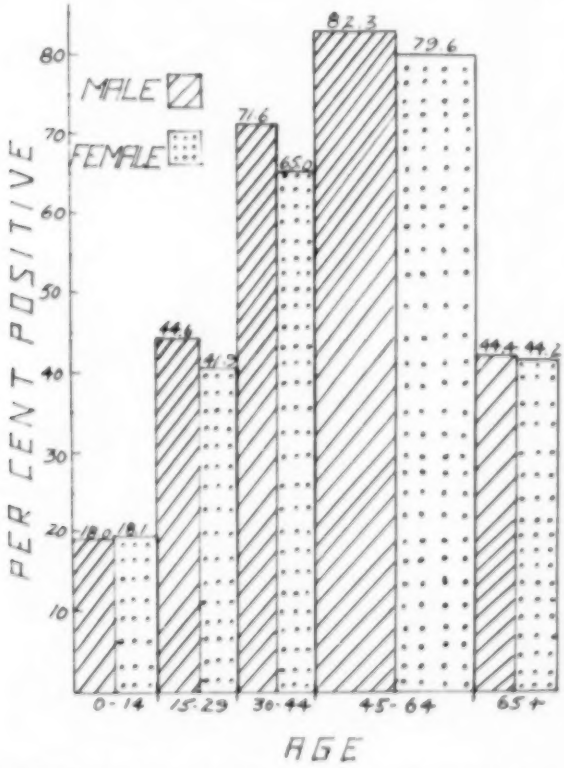

FIGURE 3: Tuberculin positive reactors by age and sex in larger age groups.

require a longitudinal study. However, it is felt that this study is useful, making due allowances for its shortcomings. There are few indices of current infection in metropolitan communities for all age groups. It was also felt that this project could be useful as a basis for a similar study of the rate of change at the next decennial census. The rate among children is probably exaggerated in the lower age group, since the rate includes contacts whose rates are higher than non-contacts in this age group. It is also generally exaggerated by the larger proportion of non-whites in the clinic than present in the general population. It was believed that it would be advisable to apply the general clinic tuberculin rate to find the total population assumed to have a positive tuberculin reaction rather than the non-contact rate, since it would not eliminate similar groups in the general population who are in contact with known and unknown cases of tuberculosis. That this group is probably not inconsiderable, is shown by the fact that on December 31, 1949 there were known to be registered 1,057 cases of tuberculosis in this community. The number of unknown cases is probably at least as large.

The method employed using a single intermediate test dose of 0.1 cc. of 1:1000 Old Tuberculin instead of the usual method of double testing

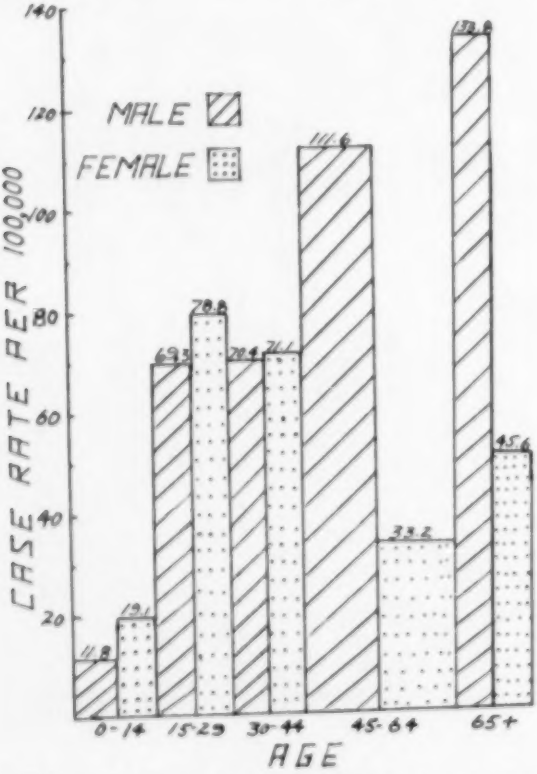

FIGURE 4: Tuberculosis case rate per 100,000 population by age and sex.

with weak and then strong doses was preferred in order to eliminate the large number of non-specific reactions which occur on testing with the large dose. This has been shown by others.[2] Our procedure is the method recommended for survey work in Diagnostic Standards, 1950 edition, of the National Tuberculosis Association.

Comparison of findings with other metropolitan studies is of some interest. The most recent study, similar to ours, was that in Rochester, N. Y., 1943-1946 by Beaven.[3] The Vollmer patch test was used on hospital children and the adults were industry groups. The differences from our study are attributable to the different locales and dates and to the fact that we used Mantoux tests which we considered more sensitive than the Vollmer patch test. The results differ particularly in children where our tuberculin rate is higher with a much lower case and death rate. Beaven's survey also showed a considerable sex difference in tuberculin reactions in adult life which was not found in our community. Important similar findings are the preponderance of new disease and death from tuberculosis in older males. Both studies show a similar falling off of tuberculin rates among older individuals.

There is no similar previous study in New York City in recent years. For comparison, we have available an unpublished study of Vollmer patch

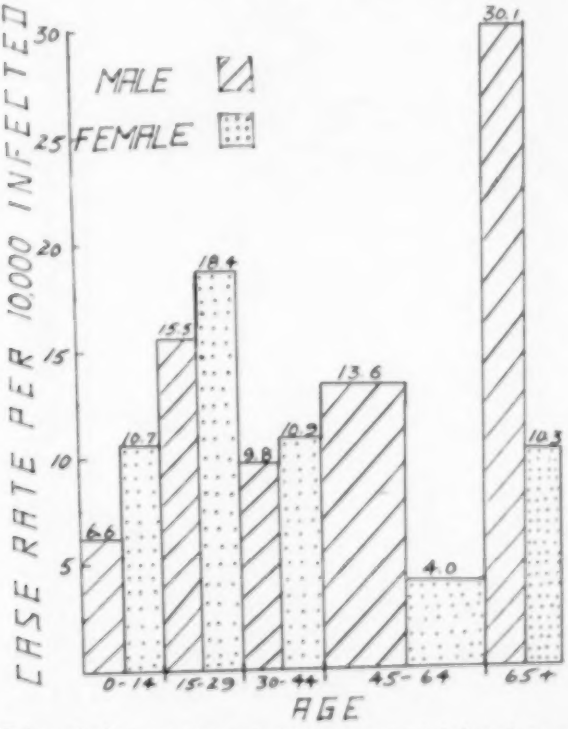

FIGURE 5: Tuberculosis case rate per 10,000 assumed infected population by age and sex.

testing done by the Queensboro Tuberculosis and Health Association from December, 1950 to May 1951 in schools in Queens County, New York City which includes the Jamaica district. Findings here were 9.5 per cent positive in the 13 to 19 age group. In 1940, Robins[4] made a tuberculin survey of New York City high school children. The group of average age, 16 to 17, had 54.5 per cent positive tuberculin reaction.

SUMMARY AND CONCLUSIONS

1. A tuberculin testing survey was made in 1949 and 1950 of all admissions to a New York City chest clinic. Tests were administered to 5,209 persons.

2. The tuberculin reactor rate increased steadily in this community to a maximum at 45 years of age. There was no apparent difference in the male and female rates. The rates among non-whites were consistently higher than among whites.

3. There was a falling off in the tuberculin reactor rate in the older age group possibly due to decrease of skin sensitivity. However, the test is useful because of large case and death rates occurring in this age group in those with positive reactions.

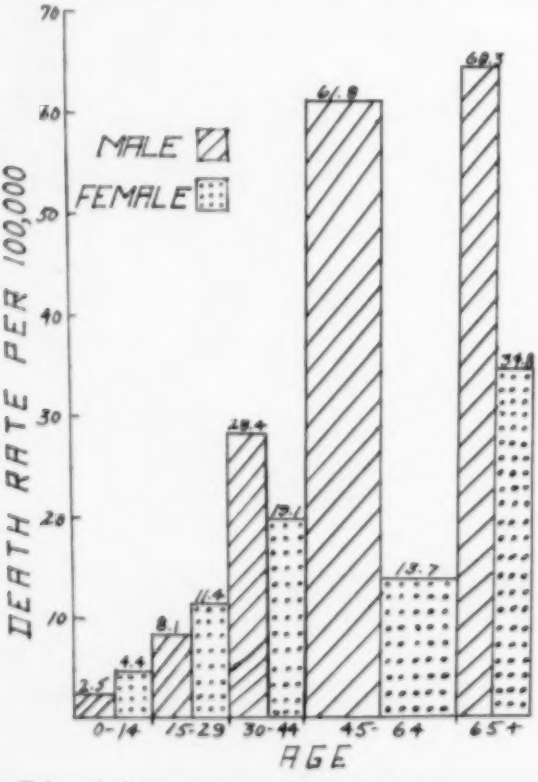

FIGURE 6: Tuberculosis death rate per 100,000 population by age and sex.

4. The tuberculin reaction rate among non-contacts is lower than that among contacts in childhood and adolescence; but by adult life there is no apparent difference in contact and non-contact rate. This is believed due to the general tuberculinization of the population by contact with known and unknown cases of this disease.

5. In this community, more new cases of tuberculosis occur in female than in male children, and rates for both sexes rise abruptly in adolescence. The maximum female case rate occurs from 15 to 29, but the male rate continues to rise through middle life and old age until the rate of new cases is more than three times that of females. This is true both in the general population and in those infected as shown by positive tuberculin tests.

6. Though non-whites comprise only 6.4 per cent of the population, they contribute 22.5 per cent of new cases.

7. Deaths are few from tuberculosis in children and adolescents in this community. The maximum female death rate is reached from 30 to 45 years. The male death rate rises continuously from birth to old age,

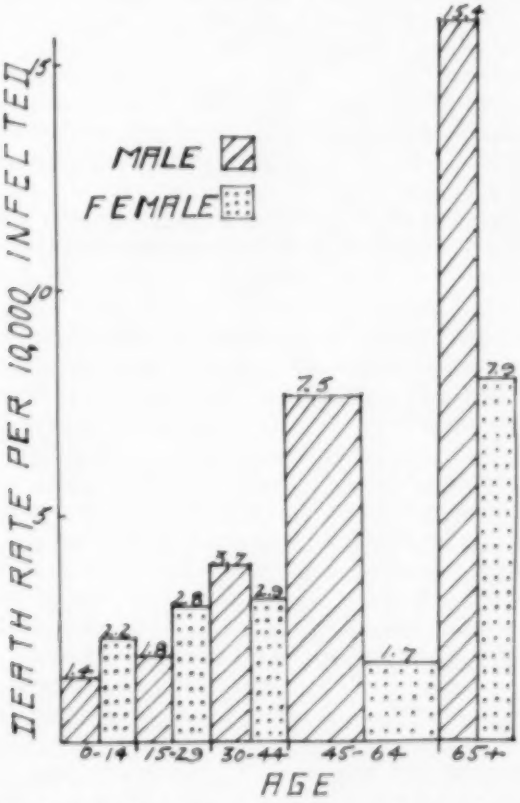

FIGURE 7: Tuberculosis death rate per 10,000 assumed infected population, by age and sex.

and from 45 through 64 is four times the rate among females. The large excess of the male death rate over that of females is found both in the general and infected population.

8. In this community, prevention of clinical disease and death from tuberculosis should be concentrated on the older male. However, there is a large potential reservoir of tuberculous infection from adolescence onward as shown by the tuberculin reactor rate. Only when this rate falls to a low level would one consider that the tuberculosis problem was on the way to solution. The chief seed bed for this high tuberculin reactor rate is probably in the known and unknown older males with tuberculosis who constitute a large reservoir of cases and are the source of the high death rate.

8. Special emphasis should be given to the non-white population who contribute a marked excess of known new cases and of deaths over their ratio in the general population.

10. The high tuberculin rate found in Jamaica, N. Y., suggests that there may be a reservoir of unknown tuberculous cases causing a general tuberculinization of non-contacts in the population by adult life, though in the absence of a longitudinal study, the high rates in adult groups may reflect the more general infection earlier in this century.

RESUMEN

1. Se llevó a cabo una prueba tuberculínica en 1949 y 1950 entre todos los casos admitidos a las clínicas de tórax de New York, Estas pruebas se hicieron a 5.209 personas.

2. La reactividad a la tuberculina ascendió en esta comunidad hasta el máximo a los 45 años de edad. No hubo aparente diferencia segúnlos sexos. La proporción entre los blancos y no blancos, fué francamente más alta entre estos últimos.

3. Hubo un decrecimiento de la reactividad a la tuberculina en el grupo de edad mayor posiblemente debido a un decrecimiento de la sensibilidad cutánea. Sin embargo, la prueba es útil a causa de la proporción elevada de casos y defunciones entre los de edad avanzada con reaciones positivas.

4. La reactividad a la tuberculina entre los sin contacto, es más baja que entre los contactos en la niñez y en la adolescencia, pero en la edad adulta no hay diferencia entre los sin contacto y los que lotienen.

5. En esta comunidad ocurren más casos de tuberculosis entre las niñas que entre los niños y la proporción se eleva bruscamente en la adolescencia. El máximo entre las mujeres se presenta entre 15 y 29 años, pero entre los hombres continúa ascendiendo a través de la edad media de la vida y la senectud hasta que la proporción de nuevos casos es más de tres veces mayor que en las mujeres. Esto ocurre tanto en la población general como en los infectados, como lo muestra la reacción tuberculínica.

6. Aunque los no blancos comprenden sólo el 6.4 por ciento de la población, proporcionan el 22.5 por ciento de los casos nuevos.

7. Las muertes por tuberculosis son escasas entre los niños y los adolescentes. El máximo de la mortalidad entre las mujeres está entre 30 y 45

años. La mortalidad entre los hombres, desde el nacimiento hasta la senectud, sube gradualmente y de los 45 a los 64 años, es cuatro veces mayor que la de las mujeres. El gran exceso de mortalidad entre los hombres sobre las mujeres, se encuentra tanto en la población general como en los infectados.

8. En esta comunidad la prevención de la enfermedad clínica y la muerte por tuberculosis, debe concentrarse en el hombre de edad. Sin embargo, hay un reservorio potencial de infección tuberculosa desde la adolescencia en adelante, según lo muestra la proporción de reactores.

9. Debe ponerse especial atención en la población no blanca, que contribuye en considerable exceso de nuevos casos y muertes en relación a su proporción en la población general.

10. La alta proporción encontrada en Jamaica, New York, sugiere que puede haber un reservorio de casos desconocidos que causan una tuberculización de no contactantes en la población adulta, aunque en ausencia de un estudio longitudinal las altas incidencias en los grupos de adultos puede reflejar una infección general más temprana en este siglo.

RESUME

1. Une recherche de l'allergie tuberculinique systématique fut réalisée en 1949-50 pour tous les malades admis dans une clinique pulmonaire de la ville de New-York. Ainsi furent testées 5.209 personnes.

2. Dans cette collectivité, la proportion des individus allergiques s'accrut d'une façon constante en fonction de l'âge jusqu'à 45 ans. Il n'y eut pas de différence notable dans les groupes d'hommes et de femmes. Les moyennes furent nettement plus élevées parmi ceux qui n'appartenaient pas à la race blanche.

3. Dans un groupe de gens plus âgés il y eut une chute du nombre des individus allergiques dont la cause est probablement une diminution de la sensibilité cutanée. Toutefois, la réaction tuberculinique est utile dans ce cas à cause de la fréquence des cas de tuberculose, et de la mortalité chez les gens âgés à réaction positive.

4. Dans l'enfance, et dans l'adolescence, la moyenne des réactions tuberculiniques est plus basse chez les individus qui ne sont pas en contact avec les tuberculeux que chez ceux qui sont en contact. Mais chez les adultes, il n'y a pas de différence apparente pour ceux qui sont en contact ou pour les autres. Les auteurs pensent que cette constatation est en rapport avec la tuberculinisation générale de la population, acquise par contact avec des tuberculeux connus ou ignorés.

5. Dans la collectivité étudiée, il y eut plus de nouveaux cas de tuberculose chez les enfants du sexe féminin que chez les enfants du sexe masculin, et la moyenne s'élève rapidement au cours de l'adolescence. La proportion maximum des cas féminins survient entre 15 et 19 ans, mais la proportion chez les hommes continue à s'élever au cours de l'âge moyen de la vie, et même de la vieillesse jusqu'à être à ce moment trois fois plus élevée que chez les femmes. Cette constatation est valable autant dans l'en-

semble de la population que chez ceux qui sont connus comme ayant une réaction tuberculinique positive.

6. Bien que les individus n'appartenant pas à la race blanche ne représentent que 6,4% de la population, ils entrent pour 22,5% dans la proportion de nouveaux cas découverts.

7. Il y a peu de morts dues à la tuberculose chez les enfants et chez les adolescents dans cette collectivité. La proportion maximum de mortalité féminine s'est localisée entre 30 et 45 ans. La mortalité augmente continuellement depuis la naissance jusqu'à la vieillesse chez les hommes, et chez ceux de 45 à 64 ans, la mortalité est quatre fois supérieure à celle des femmes. Cette proportion beaucoup plus considérable de la mortalité masculine a été constatée aussi bien dans l'ensemble de la population que chez ceux qui réagissent à la tuberculine.

8. La prévention de la maladie tuberculeuse et de la mort doit être surtout exercée dans cette collectivité chez les hommes d'un certain âge. Toutefois, la recherche des réactions tuberculiniques montre qu'il existe un large réservoir d'infection tuberculeuse à partir de l'adolescence.

9. Il faut insister tout spécialement sur les individus du sexe masculin et non de race blanche, pour lesquels les cas nouveaux de tuberculose et les cas de mort dues à cette affection dépassent de beaucoup les chiffres constatés pour l'ensemble de la population.

10. La proportion élevée d'individus allergiques qui a été constatée dans le district de Jamaica (New-York) permet de penser qu'il existe un réservoir de tuberculeax méconnus que est la cause d'une tuberculinisation généralisée parmi les individus adultes qui ne sont pas en contact avec des malades. La proportion plus élevée chez les adultes est peut-être en rapport avec une plus grande extension de l'infection qu'au début de ce siècle.

ACKNOWLEDGMENT:

We gratefully acknowledge the help of the Statistical Division of the Bureau of Tuberculosis of New York City for the statistical material.

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The Spread of Pulmonary Tuberculosis: A Vascular Pressure Hypothesis

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The purpose of this hypothesis is to analyze some of the circulatory phenomena, sometimes associated with tuberculosis, as a problem in hydrodynamics and to endeavor to confirm this analysis with known clinical and postmortem findings in hypertension, mitral stenosis, active congenital pulmonary stenosis and the behavior of tuberculosis when present during pregnancy.

It is hoped it may be possible to relate some of the observations made by Wearn et al.[1] on blood movements in the capillary circulation to the histological findings of Wm. Snow Miller[2] in those areas of the lung where the systemic and pulmonary circulation anastomose; to speculate what might occur if any pathological or congenital condition should disturb these relationships; and to relate these speculations to the postmortem x-ray findings of Wood and Miller.[3]

Miller[2] found it was in that area in the respiratory bronchiolus where the bronchial artery and the pulmonary circulation anastomose that tuberculosis tends to develop. This was true both in human and in experimental tuberculosis. He gave no certain reason for this but speculated that it might in some way be due to retardation of the circulation in this area.[2] That this impressed him is seen in that he comes back to this in his chapter on what he considers are the important anatomical areas in the lung, Key Points.[2]

Reversal of Flow in the Arterioles and Capillaries of the Lung

The studies of Wearn et al.[1] may furnish a clue to the probable mechanism in the normal lung wherein the transitional venous capillary area, mentioned by Miller[2] may receive part, at least, of its arterial blood supply. This hypothesis will attempt to show how the breaking down of this capillary area may be responsible for the spread of pulmonary tuberculosis to these and nearby areas; how, due to certain congenital defects which result in circulatory hypertension, the advance of the disease is retarded or, due to defects which result in this hypotension, the course of the disease is accelerated; and how, in the normal lung, the effects of these forces are cumulative.

In a direct microscopic study of the living mammalian lung while this was still within the unopened thorax, Wearn[1] found that the flow of blood in the arterioles and the capillaries might be intermittent, pulsatile and might even reverse its direction of flow and this reversal of direction, once started, might continue for as long as two minutes. "And we believe it to be the normal behavior of these vessels."[1]

When we study the transitional area mentioned by Miller[2] Fig. 1, we observe that the arterial blood in the bronchial artery is moving distally in the respiratory bronchiolus. It is here that the bronchial artery anastomoses with the capillaries from a root of the pulmonary artery containing venous blood coming from the opposite direction. The capillaries from this pulmonary artery, carrying venous blood, in turn anastomose with the capillaries from a root of the pulmonary vein. Figure 2 is an attempt to indicate diagrammatically the fluid forces which may be at work in this area.

A study of this area described by Miller[2] will show that nature has not provided a separate arterial blood supply. Miller definitely shows this area to have a venous blood supply. There are, therefore, only two ways in which this area can receive its arterial supply:

1. By the forward movement of the arterial blood in the bronchial arteries displacing the venous blood from the pulmonary artery; or
2. By the backward and forward movement in the capillaries and arterioles from one of the roots of the pulmonary vein[2] in the manner described by Wearn[1]; or by a combination of both.

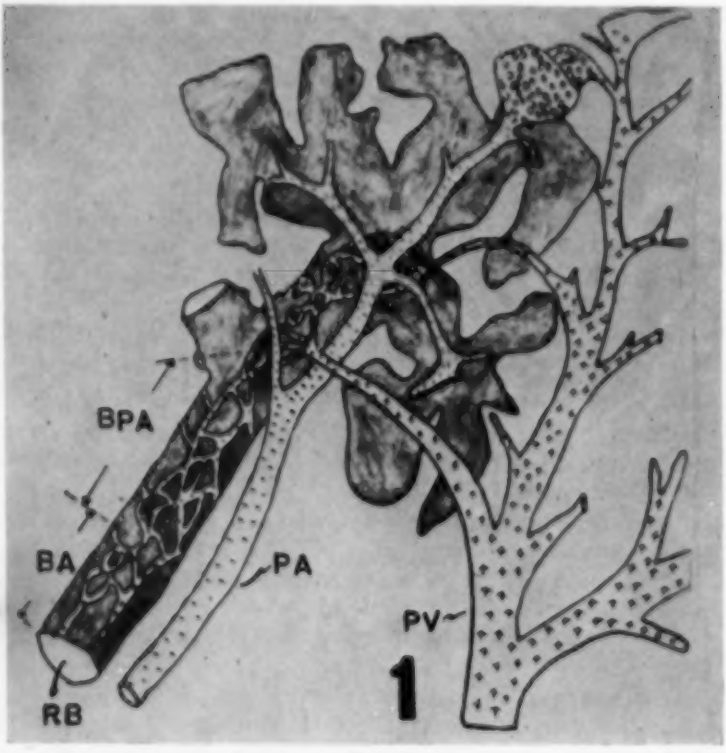

FIGURE 1

BPA—Bronchial-Pulmonary Anastomosis
BA—Bronchial Artery
PA—Pulmonary Artery
RB—Respiratory Bronchiolus
PV—Pulmonary Vein
+—Arterial Blood
—Venous Blood
⧺—Capillary Anastomosis

Since clinical findings fit these two mechanisms they have been applied to the development of this hypothesis.

Figure 2 indicates diagrammatically the forces present in the area mentioned by Miller.[2] The arterial blood is indicated by the plus (+) sign, the venous blood by the minus (—) sign, the capillary anastomosis by crosshatching. The arrows indicate the relative amounts of blood from the bronchial artery which reach the pulmonary artery and vein (Wood and Miller[3]).

In this area the pressures in the bronchial artery and the pulmonary circulation in the normal lung, due to the capillaries' resistance, must be fairly well in fluctuating equilibrium, otherwise the circulatory system, having the greater pressure, would force its blood into the adjoining system and eliminate it as a separate entity. According to Wood and Miller, there is a small amount of blood normally reaching the lungs from the bronchial arteries.[3] Since in this area the capillaries are shown as being venous[2] and since a deficiency in the arterial blood supply results in a favorable condition to support the growth of tubercle bacilli,[4] it would follow that any condition which would interfere with the supply of arterial blood to this area, such as hypotension, either systemic or pulmonary, would facilitate the growth of tubercle bacilli. Any change that would tend to break down the capillary structures in this area would allow new forces to come into play. This breaking down of the capillaries could conceivably be accounted for by the growth of tubercle bacilli.

Results of the Breakdown of the Bronchial-Pulmonary Anastomosis in Pulmonary Tuberculosis

Wood and Miller[3] perfected a postmortem method for visually demonstrating the anatomic changes which occur in the dual circulation of the lung in tuberculosis, cardiac lesions, etc. They showed that, while in the normal lung it was impossible with their method to demonstrate a direct connection between the bronchial and pulmonary circulations,[3] it was possible to show that such a direct anastomosis does exist in chronic pulmonary tuberculosis. And it seems that the only way in which this direct anastomosis can be explained is through the adverse effects upon the capillaries, either by the tuberculous process or through some pressure disturbance. With the destruction of the capillaries, the flow of blood would be from the systemic to the pulmonary since the pressure in the bronchial arteries is greater than in the pulmonary circulation. To quote them directly,

"The injection studies indicate quite clearly that an increase in the supply of oxygenated blood occurs in and about the diseased area" and

"In some cases this increase must have been enormous."[3]

These statements would seem to be in direct conflict with that of Dock.[4] He has furnished evidence that there is a deficiency of arterial blood in the apices when the patient is in an upright position and it is in this resulting medium that the tubercle bacilli flourish.[4] However, Wood and Miller[1] have visually demonstrated that in chronic pulmonary tuberculosis

there is an ample amount of oxygenated blood reaching the apices to foster maintenance of normal local conditions. In spite of this increased arterial blood supply there is no evidence that this contributes to eventual improvement of the patients. In fact, all evidence points to the contrary.

It was in order to consider the fluid forces which might be responsible for these seemingly contradictory conclusions that the diagrams shown in Figs. 3 and 4 were set up.

Figure 3 represents diagrammatically the fluid forces present in two respiratory bronchioli. Only two are shown although any number more than one could be used to demonstrate the fluid forces which are present in this area. In this particular diagram, should any pathology or pressure forces, or a combination of these, dilate or obliterate one of the capillary anastomoses between the bronchial and pulmonary circulation, we would have a situation as shown in Fig. 4.

Here the capillary anastomosis, (Fig. 4), has been obliterated and, as the result, we have a direct shunt between the bronchial and pulmonary circulations. The two systems are now directly connected and the bronchial artery empties its flow, without capillary resistance, into the pulmonary vein and on into the left auricle.

The following are the reasons why the flow would go to the pulmonary vein rather than to the pulmonary artery; First, there is a wider anastomosis between the bronchial artery and the pulmonary vein than between the bronchial artery and the pulmonary artery. Three times as much bronchial arterial blood reaches the vein as reaches the artery.[3]

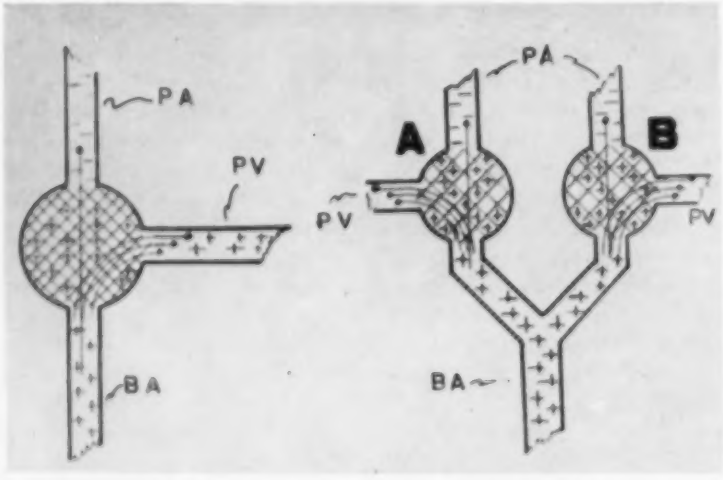

FIGURE 2

BA—Bronchial Artery
PA—Pulmonary Artery
PV—Pulmonary Vein
—Venous Blood
+—Arterial Blood
ꝯ—Capillary Anastomosis

FIGURE 3

BA—Bronchial Artery
PA—Pulmonary Artery
PV—Pulmonary Vein
—Venous Blood
+—Arterial Blood
ꝯ—Capillary Anastomosis

Second, the pressure in the pulmonary vein is less than that in the bronchial artery and the direction of flow would be to the point of lesser pressure. Since there is so great a difference in the pressure between the bronchial artery and the pulmonary circulation, the volume of arterial blood flowing through a direct anastomosis of this nature, when of long standing, must, as demonstrated by Wood and Miller,[3] be enormous.

However, it is what now occurs in the nearby respiratory bronchioli as the result of this break-through which is significant: (Fig. 4); for it is this change in the vascular system which may be responsible for changing the area from a predominantly arterial to a more venous medium and so furnishing a more favorable medium for the growth of the tubercle bacilli. This increased flow from the bronchial artery to the pulmonary circulation must result in a lowering of the pressure in the bronchial-pulmonary anastomosis of the nearby respiratory bronchioli. This must be true since there is now less capillary resistance to dam the flow in the bronchial arteries and so maintain the systemic arterial pressure.

An analogy would be a domestic water supply. If there is water running from one faucet in the house, the turning on of another faucet or the breaking of a pipe would lower both the pressure and the amount of water being delivered to the original faucet.

This lowering of pressure will therefore, result in a smaller volume of blood from the bronchial artery reaching the nearby uninfected bronchial-pulmonary anastomoses. When these break-throughs become sufficiently extensive and the pressure of the bronchial artery in the healthy capillaries falls below the pressure in the pulmonary artery, we have a condition as shown in Fig. 4. Here the pressure in the bronchial artery has fallen below that in the pulmonary circulation, as shown by the height of the four arrows. Since the pulmonary artery has greater pressure than the pulmonary vein, venous blood rather than arterial blood would move into the capillary area. Since venous blood furnishes a more favorable medium for the growth of the tubercle bacilli,[4] we probably have a destructive process occurring which would tend to further direct anastomosis between the bronchial artery and the pulmonary circulation. The process would then seem to become cumulative, each additional break-through contributing to the further spread of the disease.

It is, therefore, possible to reconcile the seemingly contradictory conclusions of Wood and Miller[3] on one hand with those of Dock.[4] In the light of this approach, their conclusions are complementary rather than contradictory, since each breakthrough of the bronchial artery into the pulmonary circulation contributes to the extension of venous areas in the surrounding respiratory bronchioli.

Effect of Pressure on the Abdominal Aorta in Pulmonary Tuberculosis

Wearn[1] demonstrated that when pressure was applied to the abdominal aorta blood appeared in the arterioles and capillaries of the lung in areas where these vessels had previously closed and were invisible. When this

pressure was removed the blood flow ceased and as the result of the constriction of the vessels, blood was squeezed out in opposite directions.

In this case, the forces present are shown in Fig. 5. Pressure on the abdominal aorta, Fig. 5, tends to develop a back-pressure in the left ventricle. This, in turn, interferes with the emptying of the left auricle and, as a result, the blood begins to back up in the pulmonary vein. Consequently, with each subsequent contraction of the left ventricle, this arterial blood in the pulmonary vein which is unable to drain into the left auricle is forced back into the capillaries in the manner described by Wearn, and, probably, into the area mentioned by Miller.[2] It is in this way that one can visualize a mechanism for furnishing an arterial blood supply for the venous area where Miller[2] found his evidence of beginning tuberculosis.

However, it is in pregnancy, complicated with tuberculosis, where we find a situation paralleling this condition, i. e. pressure on the abdominal aorta, and in pregnancy we have a substantial amount of clinical evidence which may give weight to this analysis.

In preganacy where we have a previous history of tuberculosis, we often find improvement during the period of pregnancy and a relapse after the child is born. The mechanics in such case can be reasoned to be similar to that shown in Fig. 5. The increase in size and weight of the foetus must result in increased pressure on all structures within the abdominal cavity and thereby create some degree of back-pressure in the abdominal aorta in the manner mentioned by Wearn.

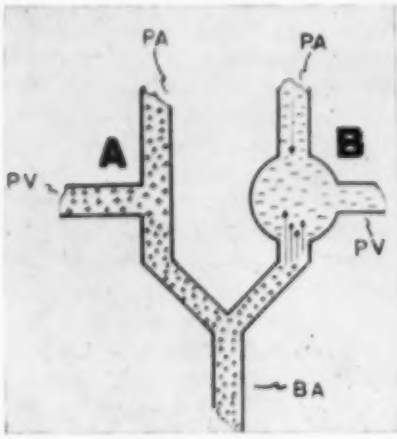

FIGURE 4

BA—Bronchial Artery
PA—Pulmonary Artery
PV—Pulmonary Vein
—Venous Blood
+—Arterial Blood
ǂ—Capillary Anastomosis

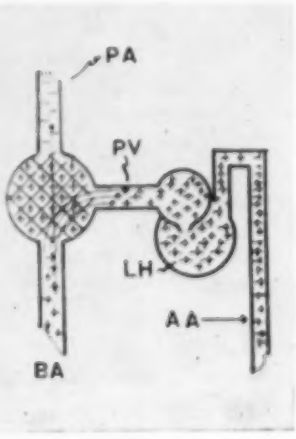

FIGURE 5

BA—Bronchial Artery
PA—Pulmonary Artery
PV—Pulmonary Vein
LH—Left Heart
AA—Abdominal Aorta
—Venous Blood
°—Arterial Blood
ǂ—Capillary Anastomosis

If it can be assumed that an ample arterial blood supply from the pulmonary circulation to the affected areas is important in recovery from pulmonary tuberculosis,[4] then this may be a possible explanation of the mechanism involved. When the postmortem findings of Wood and Miller are applied to the observations by Wearn[1] and we analyze the processes destructive to the bronchial-pulmonary capillary anastomosis due, probably, to the dilatation of the capillary bed[3] which may have occurred during the period of pregnancy, we may find a clue to the reason the tuberculous patient is worse following her pregnancy than she was before.

Therefore, it is logical to reason that if, during pregnancy, these anastomoses have been increased due to this back-pressure,[3] then, as soon as this pressure is removed, the bronchial artery, now having greater pressure than the pulmonary vein, would force an excessive volume of blood into the now dilated pulmonary vein anastomosis. This would then repeat the cycle which is explained under "results of the Breakdown of the Bronchial-Pulmonary Anastomosis in Pulmonary Tuberculosis."

The mechanism explained in Fig. 5 in modified form can also be applied to hypertension[7] and mitral stenosis in their relation to tuberculosis. However, in mitral stenosis the constriction is at the mitral valve, while in hypertension it may involve any portion of the systemic arterial circulation arising from the left heart. However, the final result would be the same, namely, increasing the arterial blood supply to the area of the respiratory bronchioli by forcing the blood backwards in the pulmonary vein. This may explain the slowing down of the tuberculous process when hypertension is present. Wood[3] furnishes us with confirmatory evidence to support this conclusion. It was found that where there was an increased pulmonary venous pressure there was a tendency for these bronchial-pulmonary artery anastomoses to develop.

Wood and Miller.[3] "In cardio-vascular disease which *resulted in heightened pulmonary pressure*, and possibly associated with lesions of the pulmonary vascular tree, numerous bronchial-pulmonary arterial anastomoses develop."
(Italics ours).

Application to Apical Tuberculosis

This hypothesis can also be used to explain the tendency of tubercle bacilli to first attack the apices of the lungs. Dock[4] has shown that the pressure in the pulmonary circulation is not sufficient to furnish an adequate blood supply to this area when the patient is in an erect position. However, Wood and Miller[3] have demonstrated that once the infection is extensive there is a more than adequate arterial blood supply to the affected area and it would appear from their x-ray film studies that the greater this vascularization the more hopeless the prognosis.

To reiterate, when the blood supply from the bronchial artery breaks through the bronchial pulmonary capillary anastomosis, the decrease in resistance due to a possible modification of the capillary bed allows an increased flow of arterial blood to pass through this direct shunt. This, in turn, reduces the bronchial arterial pressure in the adjoining respiratory bronchiolus. With this reduction of arterial pressure and blood

supply, the blood in the adjoining bronchioli tends to become venous, as previously explained; this, in turn, means a more favorable medium for the growth of tubercle bacilli. It is reasonable to suppose that the growth of these organisms would tend to destroy the capillaries in these areas. Once destroyed, the bronchial circulation breaks through in this new area and we get a further reduction in the pressure of the systemic arterial circulation. The process is cumulative.

Unlike previous explanations, this hypothesis furnishes us with an explanation for the spread of tuberculosis in all parts of the lungs where the capillary anastomosis between the pulmonary and bronchial circulations is impaired. The probable explanation of why the infection is more prevalent in the apices is apparent when we apply the mathematical observations of Dock[4] to the reversal of flow in the capillaries by Wearn et al.[1] In the erect posture there may be little circulation in the apical area; this, again, fosters a favorable medium for tubercle bacilli. This hypothesis, however, can be applied to other areas of the lung.

The same reasoning which would apply to the mechanism explaining the spread of tuberculosis in the apices can be used to explain the spread of the disease in congenital stenosis of the pulmonary orifice. Here again we would have an inadequate pulmonary blood supply; but here it would affect all parts of the lungs, the same mechanism described under "Results of the Breakdown of the Bronchial-Pulmonary Anastomosis in Pulmonary Tuberculosis. It would, therefore, be reasonable to suppose that the rate at which the infection spreads might depend upon the degree of stenosis.

One other observation may be in order here, a possible explanation why the right and not the left apex is first affected. The difference in the arterial blood supply to the two lungs furnishes another approach to the problem. While most anatomists,[5, 6] call attention to the anomalies in the origin and the number of the bronchial arteries, they generally agree that the usual number is three; and that two of these supply the left lung and one the right. Is it possible that this difference in number and, consequently, the difference in the supply of arterial blood from the systemic circulation to the two lungs may be in part responsible for the tendency of the tubercle bacillus to find a more favorable medium for its growth in the right lung than in the left?

SUMMARY

This hypothesis attempts to explain (1) the spread of pulmonary tuberculosis as a vascular pressure phenomena. (2) Why pulmonary tuberculosis first appears in the area of the pulmonary broncheal anastomosis. (3) Why extensive infection in this area results in lowering the arterial pressure. (4) why when mitral stenosis is present it results in the slowing down of the tubercular process. (5) Why during preganacy, pressure on the abdominal aorta results in the improvement of the patient and why after delivery, this improvement ceases. (6) A possible explanation of why the right lung is attacked first in apical tuberculosis. (7) The

part these vascular forces play when congenital stenosis of the pulmonary orifice is present.

RESUMEN

Esta hipótesis pretende explicar (1) la diseminación de la tuberculosis pulmonar como un fenómeno de presión vascular. (2) Por qué la tuberculosis pulmonar aparece primero en el área de las anastomosis pulmonares-bronquiales. (3) Por qué la infección extensa en esta área da como resultado un descenso de la presión arterial. (4) Por qué cuando se presenta la estenosis mitral resulta la lentitud del proceso tuberculoso. (5) Por qué durante el embarazo la presión sobre la aorta abdominal acarrea la mejoría de la enferma y por qué después del parto esta mejoría cesa. (6) Una explicación de por qué el pulmón derecho esatacado primero en la tuberculosis apical. (7) El papel que estas fuerzas vasculares desempeñan cuando hay estenosis congénita del orificio pulmonar.

RESUME

L'auteur tente de rendre compte:

1) De l'extension de la tuberculose pulmonaire par un phénomène de pression vasculaire; 2) d'expliquer pourquoi la tuberculose pulmonaire apparait d'abord au point où se fait l'anastomose broncho-pulmonaire; 3) pourquoi l'extension de l'infection dans cette région est due à l'abaissement de la pression artérielle; 4) pourquoi l'existence d'une sténose mitrale permet de freiner l'extension du processus tuberculeux; 5) pourquoi au cours de la grossesse, la pression de l'aorte abdominale a pour conséquence l'amélioration de la malade, et pourquoi aprés la délivrance, cette amélioration cesse; 6) Il donne une interprétation possible de la raison pour laquelle le poumon est d'abord atteint de tuberculose au niveau de son sommet; 7) il explique quelle part ces phénomènes vasculaires jouent en cas de rétrécissement congénital de l'orifice de l'artère pulmonaire.

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7 Smart, Reginald H., Los Angeles, Calif. In conversations with Dr. Smart, he called my attention to his observations over an extended period covering hypertension in pulmonary tuberculosis. He observed that while these patients never were cured of their tuberculosis, they usually died of some other ailment.

Bilateral Resection Therapy in Pulmonary Tuberculosis*

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Treatment of bilateral pulmonary tuberculosis has always been difficult when strict bed rest supported by drug treatment has not led to recovery.

In most cases clinical cure of the process occurs in one of the lungs after a certain period of time, but usually there remains a residual unilateral active process, which may be dealt with by collapse or resection therapy.

For many years bilateral collapse therapy has been used in the form of intrapleural or extrapleural pneumothorax in bilateral processes still showing signs of activity. A satisfactory result, including recovery of the lung function, may be obtained when the course of intrapleural pneumothorax is free from pleural complications. We know, however, that mild pleuritis or even an increased deposition of fibrin on the pleura may give rise to considerable reduction of the ventilation reserves.

The same disadvantage is involved in extrapleural pneumothorax, in which, both during the institution of it and after the termination of the refillings an exudate is frequently observed.

Bilateral thoracoplasty is only rarely carried out, because the loss of function is too great, especially following resection of a large number of ribs. Moreover, the chances of good results of bilateral collapse therapy are restricted to pulmonary tuberculosis with cavitation only, because caseous foci with or without liquefaction, bronchiectasis and tuberculous bronchitis in general remain uninfluenced.

These patients will only be cured by resection of the diseased tissue. This is of great importance for a group of tuberculosis patients, who from time to time expectorate sputum containing few bacilli in bilateral processes showing little activity. These patients are usually not ill in the clinical sense, but, owing to their positive sputum, they must be deferred from many social activities. These seemingly inactive abnormalities may cause a reactivation or dissemination with grave consequences, because then it is often too late for active therapy of whatever form.

When the localization of the abnormalities is well demarcated and when the operation is not accompanied by too great a loss of functioning lung tissue, resection will be possible, and, when there are no complications, this certainly need not cause serious loss of pulmonary function.

It goes without saying that the pre-operative examination for the localization of the affection must be carried out as accurately and fully as possible, and that we must also know the whole course of the disease as accurately as possible, in order that no unpleasant surprises arise during

*From the "Beatrix Oord Sanatorium."

the operation, leading to its having to be extended further than was the intention when considering the diagnosis. For dealing effectively with incipient complications it is also necessary that the bacilli are still sensitive to the antibiotics, streptomycin in particular.

Of paramount importance, however, is the examination of pulmonary function, both before and after the first operation and following the second one. An objective evaluation of the result is only possible on the strength of the findings of these examinations.

So far little mention has been made in the literature of bilateral resection therapy.

Ryan[1] discusses a number of patients with scattered caseous foci in both lungs, in whom he applied this method of treatment. In order to spare as much lung tissue as possible, he usually removed wedges, a subsegment or a segment. The course was favourable in all patients and the cure was uneventful.

Bickford[2] *et al.* carried out bilateral resection in 10 patients. They performed a supplementary operation, which reduced the thoracic cavity, just as in many cases of unilateral operations. They usually performed phrenic crush on one side and limited thoracoplasty on the other. Unilateral lobectomy was done in three of the 10 cases, in all others, segmental resection. These authors also point out that it is of great importance to study as accurately as possible the whole course of the disease, in order not to be faced with disagreeable surprises later on, for example in the form of an unknown, seemingly cured process in a spared part of the lung, which shows signs of activity again.

Personal Investigation

We did bilateral resection in 14 patients, in all of whom the course was favourable. In all cases the affection was in general confined to at most two segments of each lung, so that it was to be expected that the function would not be influenced too strongly.

A general rule was that operation was carried out primarily on the side on which, in view of the extension and localization of the process, there was the least risk of loss of function. Should a complication arise in the second operation, then the danger for the patient was as small as possible.

The first six patients, who have already left the sanatorium some time, and who have resumed their occupations again, will be discussed extensively. The surgeon was Prof. L. D. Eerland, the anaesthetist Dr. C. R. Ritsema Van Eck, while the examination of the lung function was carried out by Dr. P. J. Van Oostrum.

All operations took place under the protection of streptomycin, which was also administered for about a fortnight post-operatively. The patients had further a post-operative bed rest course of four months, while the duration of the whole after-treatment in the sanatorium was about eight months. They had also a PAS course during the first two months following the operation.

We attach great significance to adequate after-treatment in the sana-

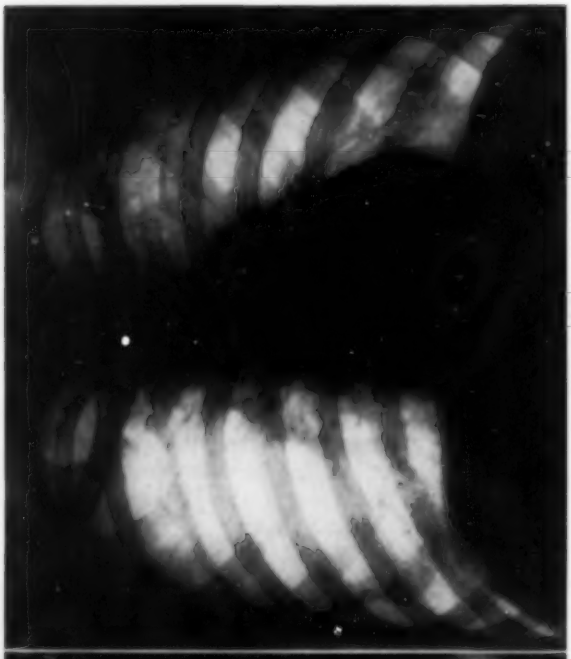

FIGURE 2

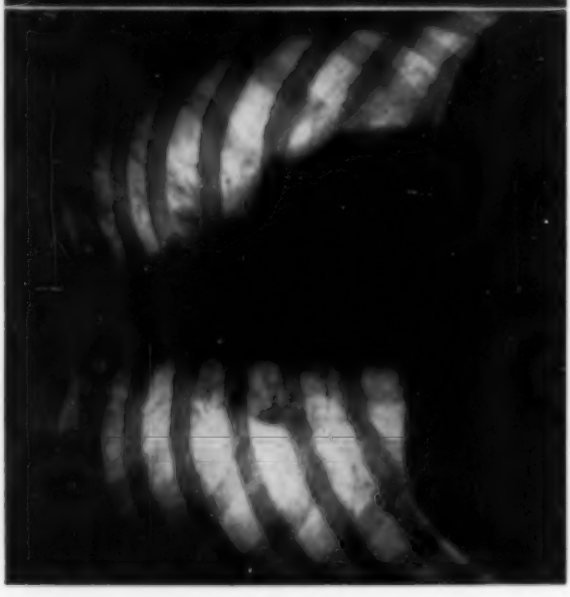

FIGURE 1

torium. This will undoubtedly exert a favourable influence on the immunological relationships, and it should always be borne in mind, even when dealing with the mildest affections, that tuberculosis is a disease which is usually not confined to one organ or part of an organ.

The examination of pulmonary function was as extensive as possible, both before the first and the second operations, and six months after the second one. The pre-operative determinations afforded the most important data for judging the possibility of an operation, while the post-operative examination constituted a valuable aid to assess the patient's fitness for work.

In the spirographic examination the determination of the vital capacity (V.C.) is not sufficient, because it neither gives an impression of the essential respiratory reserves nor of the degree of bronchospasm or emphysema of the lung. The determination of the respiratory rate according to the principle of Tiffeneau gives an important indication of the size of the reserves which will increase the residual value of the ventilation. It may be used to evaluate the influence of the bronchospastic factors. Tiffeneau spoke of the *capacité utilisable pro secunde* (C.U.S.).

The maximum respiratory minute volume (M.R.M.V.) was determined by the methods described by Hirdes[3] and Van Der Drift.[4] The results can be judged better by recording respiratory frequency of 30-35/min.

A satisfactory ventilation of the alveolar air will take place with this frequency, as is shown by the determination of the functional residual volume (F.res.det.), which in normal cases remains at least 30-40 per cent of the total capacity.

Naturally the determination of the residual volume (res.) is of importance for the judgment of emphysematous changes.

The quotient $\frac{\text{M.R.M.V.}}{\text{V.C.}}$ (normally about 20) gives a good impression of the ventilation reserve, while the respiration-equivalent (r.eq.) $\left(= \frac{\text{respiratory minute volume in liters}}{\text{oxygen uptake in ml.}} \times 100\right)$ indicates the efficiency of the respiration of both lungs together and of each lung separately. The higher this quotient, the less favourable the ventilation.

The share of each lung separately in vital capacity, R.M.V. and oxygen uptake is determined by bronchospirometry, also the determination of the equivalent.

Patient A, a 23-year-old soldier, showed on fluoroscopy abnormalities in both lungs in August 1948. The processes became more quiescent and the sputum usually became negative following a course of bed rest, supported by drug treatment. In April, 1950, a fairly large caseous focus was observed in the apex of the right upper lobe, and a number of small, rather sharply circumscribed caseous foci in the left upper lobe, while there were also a number of small foci scattered through both lungs.

In February, 1951, he was again admitted to the Military Hospital, because the sputum was positive from time to time. The abnormalities in both lungs had remained unchanged; the apical segment of the right upper lobe showed a caseous focus the size of a chestnut with a small area of liquefaction, while the apicoposterior segment of the left upper lobe and the apical segment of the left lower lobe contained a number of small foci, partly confluent (Fig. 1).

Resection was considered, and because the secretion removed with the METRAS tube from the right upper lobe was positive and that from the left upper lobe negative, it was decided to operate first on the right side.

FIGURE 4

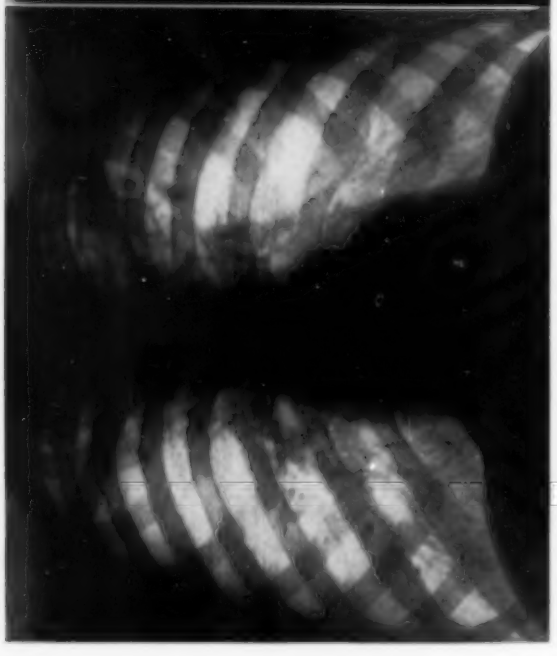

FIGURE 3

The pulmonary function examination (*Table* I-II) did not yield any contra-indications and the apical segment of the right upper lobe was removed on March 27, 1951.

TABLE I
SPIROGRAPHY

Date	2.3.'51	31.8.'51	7.3.'52	Normal
V.C.	5750 c.c.	5100 c.c.	4800 c.c.	4600 c.c.
M.R.M.V.	128 L.	111 L.	90 L.	92 L.
r.eq.	3.13 L.	2.5 L.	3.7 L.	1.5-3.5 L.
$\frac{\text{M.R.M.V.}}{\text{V.C.}}$	22.4	21.7	18.7	20
C.U.S.		71 per cent	58 per cent	70 per cent
Res.	18 per cent	18 per cent	21 per cent	24 per cent
F.res.det.		40 per cent	41 per cent	40 per cent

1e op. 27. 3.51 Ap. segm. R. upper lobe

2e op. 10.10.51 Ap. post. segm. L. upper lobe + ap. segm. L. lower lobe + wedge ant. segm.

TABLE II
BRONCHOSPIROMETRY

Date	2.3.'51		31.8.51		7.3.'52	
	R.	L.	R.	L.	R.	L.
V.C.%	52	48	49	51	61	39
R.M.V.%	52	48	50	50	55	45
O_2%	52	48	50	50	63	37
r.eq.L	2.8	2.8	2.5	2.5	3.4	4.7

1e op. 27. 3.51 Ap. segm. R. upp. lobe

2e op. 10.10.51 Ap. post. segm. L. upp. lobe ap. segm. L. lower lobe edge ant. segm.

The post-operative course was uneventful, and there was an excellent expansion with a good restoration of function. It is true, the vital capacity and the M.R.M.V. decreased, but the mutual relationship between the two lungs remained practically unchanged (*Table* I-II).

The pathologist (Prof. J. J. Th. VOS) found a tuberculoma the size of a walnut with some small tuberculous inflammatory infiltrations in the surroundings. Unfortunately the sputum remained positive from time to time after this resection, so it was decided also to operate upon the left side.

On October 10, 1951, the apicoposterior segment and the apical segment of the lower lobe were resected, while at the same time a caseous focus was removed from the anterior segment by a wedge excision.

Pathological examination revealed a number of partially caseated and calcified foci in these specimens, still containing distinct granulation tissue.

The expansion of the lung after the second operation was also excellent, but a small diaphragmatic adhesion persisted (Fig. 2).

It was to be expected that this more extensive resection and the limited mobility of the diaphragm would cause a greater loss of function (*Table* I-II).

The vital capacity fell only slightly, the M.R.M.V. somewhat more, namely 20 l., while the share of the left lung in the total function had definitely decreased with respect to vital capacity and oxygen uptake. The decrease of the C.U.S. values is perhaps a result of the diaphragmatic adhesions. The residual volume remained unchanged after the second operation also, so that there was no question of emphysema.

In view of the fact that the function of the right lung was clearly improved after the second operation, it is by no means impossible that a further recovery of the left lung will occur.

He is ambulant at present and has no particular complaints during his work.

Patient B, a female, age 29 whose abnormalities in the upper lobes of both lungs were observed in a mass x-ray film examination on May 19, 1949. A bed rest course, supported by PAS, did not lead to improvement, and she was admitted to the sanatorium in August, 1950.

A number of caseous foci without manifest liquefaction were found in the apicoposterior segments of both upper lobes.[5] The E.S.R. was slightly increased (16 mm.), the sputum negative. She was given a further course of rest in bed, and when it appeared that the condition was quiescent and that no bacilli were expectorated, she was mobilized gradually. The sputum was weakly positive in June 1951, and this was observed again in the subsequent months *during* a PAS course. In October it was therefore decided to resort to resection therapy, for which there was no contraindication, because the function of both lungs was excellent (*Table* III & IV).

TABLE III
SPIROGRAPHY

Date	16.10.'51	24.3.'52	22.10.'52	Normal
V.C.	3450 c.c.	3165 c.c.	2865 c.c.	2900 c.c.
M.R.M.V.	86.1 L.	85.5 L.	70 L.	58 L.
R.eg.	1.8 L.	3 L	1.7 L.	1.5-3.5 L.
$\frac{\text{M.R.M.V.}}{\text{V.C.}}$	25	27	24	20
C.U.S.	84 per cent	78 per cent	74 per cent	70 per cent
Res.		17 per cent	23 per cent	24 per cent
F.res.det.		34 per cent	51 per cent	40 per cent

1e op. 12.11.51 Ap. post. segm. L. (adh. diaphr.)

2e op. 31. 3.51 post. segm. R. upp. lobe + wedge ap. segm. lower lobe

TABLE IV
BRONCHOSPIROMETRY

Date	16.10.51		24.3.52		22.10.52	
	R.	L.	R.	L.	R.	L.
V.C.%	54	46	65	35	59	41
R.M.V.%	52	48	57	43	54	46
O_2%	50	50	65	35	60	40
r.eq.L	3.4	3.1	3.3	4.3	2.8	3.7

1e op. 12.11.51 Ap. post. segm. L. (diaphr. adh.)

2e op. 31. 3.52 Post. segm. R. upp. lobe + wedge ap. segm. lower lobe

The apicoposterior segment of the left upper lobe was removed on November 12, 1951. Pathological examination (Prof. J. J. Th. VOS) revealed caseous foci with a border of granulation tissue, while there were small bronchi showing tuberculous changes.

The post-operative course was uneventful, but after some time a pleural adhesion with the diaphragm developed.

Although the sputum remained negative during the first three months following the operation, it was nevertheless decided, in March 1952, to remove the apicoposterior segment of the right upper lobe.

Determination of the pulmonary function showed that the fall of the vital capacity was only 300 ml., and that the M.R.M.V. had remained the same (*Table* III & IV). It appeared, on comparison with intermediate determinations, that these favourable values occurred especially under the influence of well-regulated respiratory exercises. The share of the left lung, especially in the oxygen uptake, had however decreased considerably more than was to be expected after a segmental resection, and this had not improved in spite of the respiratory exercises.

On March 31 1952 resection of the dorsal segment of the right upper lobe was done while at the same time caseous foci, both in the apical segment of the upper and of the lower lobe, were removed by wedge excision. Pathological examination demonstrated tuberculous granulation tissue around these foci.

The lung expanded satisfactorily, but here also there was a residual pleural adhesion to the diaphragm.

The lung function determination (*Table* III & IV), more than six months following the operation, proved that there was a slight loss of vital capacity and a somewhat greater loss of the M.R.M.V., while the functional residue after hyperventilation was also higher. Inexperience will undoubtedly have been of influence here, in addition to the pleural adhesions.

Bronchospirometric determination showed that the function of the right lung had been affected very little by the operation and that the share in the vital capacity and oxygen uptake had only slightly decreased.

We may therefore conclude that, apart from the pleural adhesions, a good recovery of the lung function had occurred in this case and that it was also owing to these operations that a satisfactory clinical cure was obtained.

Patient C, a female, age 31 was admitted to the sanatorium with a chestnut-sized cavity in the apex of the left lower lobe. The cavity became smaller under the influence of bed rest, but it did not disappear, so that intrapleural pneumothorax was instituted. Although the cavity changed into a caseous focus, the sputum remained positive in spite of drug treatment.

In December 1949 a fairly large caseous focus and some areas of bronchiectasis were still present in the apical segment of the left lower lobe, while the right upper lobe showed a small cavity with some caseous foci. Intrapleural pneumothorax was instituted on the right side, which was refilled until October 1950.

The cavity on the right side had by that time also changed into a caseous focus, but the sputum was positive from time to time.

In February, 1951, the patient was admitted to our sanatorium for observation in connection with possible resection therapy. We found a fairly large caseous focus and bronchiectasis in the apical segment of the left lower lobe and some smaller, partially confluent caseous foci in the anterior segment of the right upper lobe.

Bronchial secretion was aspirated from both sides by means of a bronchoscope, and because some bacilli were found in the secretion from the right upper lobe, it was decided to resect the right anterior segment. The operation was done on January 7, 1952, after lung function examination had shown that there were no contra-indications (*Table* V & VI).

TABLE V
SPIROGRAPHY

Date	28.12.51	14.5.51	27.11.52	Normal
V.C.	4029 c.c.	3745 c.c.	3955 c.c.	3100 c.c.
M.R.M.V.	83 L.	85 L.	80.4 L.	62 L.
R.eq.	1.5 L.	1.9 L.	2.1 L.	1.5-3.5 L.
M.R.M.V. / V.C.	20.5	22.8	22.1	20
C.U.S.	79 per cent	73 per cent	70 per cent	70 per cent
Res.	18 per cent	13 per cent	7.9 per cent	24 per cent
F.res.det.	40 per cent	34 per cent	38.6 per cent	40 per cent

1e op. 7.1.52 Ant. segm. R. upper lobe
2e op 26.5.52 Ap. segm. L. lower lobe

TABLE VI
BRONCHOSPIROMETRY

Date	28.12.51		14.5.52		27.11.52	
	R.	L.	R.	L.	R.	L.
V.C.%	55	45	49	51	53	47
R.M.V.%	63	37	50	50	47	23
O_2%	53	47	48	52	51	49
R.eq.L.	2	1.3	1.9	1.7	1.1	1.3

1e op. 7.1.52 Ant. segm. R. upp. lobe
2e op. 26.5.52 Ap. segm. L., lower lobe

A number of caseous foci with a narrow border of tuberculous granulation tissue were found in the operation specimen.

The post-operative course was uneventful, apart from spontaneous pneumothorax which developed a few days after the operation. The right lung expanded excellently, as is shown by the results of the function examination on May 14, 1952 (*Table* V & VI).

There was only a slight fall of the vital capacity, but an increase of the R.M.V. and the M.R.M.V. The bronchospirometric findings prove that the share of the right lung in the vital capacity and oxygen uptake had decreased but slightly after the operation, while it had become normal again in the R.M.V. The C.U.S. and the residual values were also completely normal.

In this case it was also regrettable that the sputum remained positive after the operation, so that resection of the apical segment of the left lower lobe was decided upon. This operation, carried out on May 26, 1952, was successful, and the lung expanded nicely.

The pathologist found a number of small caseous foci and a small cavity in the specimen, largely filled with caseous substance; there was tuberculous granulation tissue at various places.

The control examination of the pulmonary function, six months following the second operation, proved that there was no loss of function. The vital capacity and M.R.M.V. had not decreased. The equivalent, the C.U.S. value and the residual values proved that the quality had not suffered either (*Table* V & VI).

Bronchospirometric examination demonstrated that there was now a normal proportion between the share of both lungs in the ventilation values as well as in the oxygen uptake. In this case there was an excellent recovery after an illness of more than five years' duration, during which period bed rest, supported by drugs and collapse therapy, had failed.

TABLE VII
SPIROGRAPHY

Date	8.10.51	14.2.52	21.10.52	Normal
V.C.	3060 c.c.	2730 c.c.	2865 c.c.	3100 c.c.
M.R.M.V.	66.9 L.	80 L.	73 L.	62 L.
R.eq.	3.1 L.	3.1 L.	3.1 L.	1.5-3.5 L.
M.R.M.V. / V.C.	21.9	29.3	16	20
C.U.S.	85 per cent	88 per cent	85 per cent	70 per cent
Res.	24 per cent	22 per cent	16 per cent	24 per cent
F.res.det.	39 per cent	33 per cent	28 per cent	40 per cent

1e op. 29.10.51 Subsegm. ant. segm. R. upp. lobe + wedge ap. segm. upper lobe
2e op. 10. 3.52 Ap. post. segm. L. upper lobe + wedge ap. segm. lower lobe

Patient D, a female, age 22 because of exposure to tuberculosis, had been under the supervision of the Tuberculosis Centre for years. In May, 1949, she felt tired; pulmonary abnormalities were found in both upper lobes, for which she was admitted to hospital in November, 1949. She was treated with bed rest and a course of PAS. The foci in both lungs remained quiescent and she was mobilized to some degree but had to be admitted to the sanatorium on account of positive sputum in December, 1950.

However, the processes showed little improvement after a rest course, supported by PAS, so that resection was considered in October, 1951.

A caseous focus the size of a chestnut with central liquefaction was found in the anterior segment of the right upper lobe, while there was a second focus of the same proportions with liquefaction in the left apicoposterior segment, in addition to a few foci the size of a pea.

Pulmonary function examination did not reveal any contra-indication, and on October 29, 1951, on the right side a subsegment of the anterior segment and a wedge from the apical segment of the upper lobe were resected (*Table* VII & VIII).

TABLE VIII
BRONCHOSPIROMETRY

Date	8.10.51		14.2.52		21.10.52	
	R.	L.	R.	L.	R.	L.
V.C.%	49	51	46	54	51	49
R.M.V.%	51	49	51	49	50	50
O_2%	45	55	43	57	50	50
r.eq.L	4.8	3.8	2.8	1.9	4.7	4.6

1e op. 29.10.51 Subsegm. ant. segm. R. upp. lobe + wedge ap. segm. upp. lobe
2e op. 10. 3.52 Ap. post. segm. L. upp. lobe + wedge ap. segm. lower lobe

The specimen yielded a caseous focus the size of a cherry with much tuberculous granulation tissue and some smaller foci with little granulation tissue.

Following the operation there was initially a rather considerable formation of fibrin, but afterwards the lung cleared up satisfactorily. The sputum remained positive and the abnormalities in the left upper lobe improved but slightly.

The lung function was only slightly changed by this selective operation (*Table* VII & VIII).

The vital capacity showed a slight fall, but the M.R.M.V. had increased, while there was a slight shift between the shares of both lungs in the total function, in favor of the left lung.

On March 10, 1952, the apicoposterior segment of the left upper lobe and a wedge from the apical segment of the lower lobe were removed. There were some caseous foci in the operation specimen, with tuberculous granulation tissue scattered through the parenchyma.

The post-operative course was also uneventful. She is up and about with constantly negative sputum.

The pulmonary function has remained highly satisfactory (*Table* VII & VIII), even though the M.R.M.V. decreased somewhat following the second operation, but its value is still higher than before the operation and it exceeds the average normal value. All other results of the examination of the pulmonary function are excellent and there is no question of emphysema. After the second operation the mutual relationship of the lungs was changed in that there was a decrease of the share of the left lung in vital capacity and oxygen uptake, but the values are at present practically normal, so that in this case also we may speak of satisfactory recovery in every respect. (Fig. 4)

Patient E, a male, age 21, in January, 1946 developed pleurisy on the right side, for which he was treated until October, 1947. He felt well until the spring of 1950 when he began to cough and in July, 1950, cavities were found in both upper lobes.

The condition improved under the influence of bed rest and drug treatment, but the sputum remained positive. On December 1951 the apicoposterior segment of the right upper lobe showed a cavity the size of a cherrystone and some small caseous foci. In the corresponding segment on the left a cavity the size of a hazelnut, also with caseous foci was found.

TABLE IX
SPIROGRAPHY

Date	27.11.51	26.3.52.	17.10.52	Normal
V.C.	3945 c.c.	3700 c.c.	3800 c.c.	3500 c.c.
M.R.M.V.	83 L.	81.8 c.c.	80 L.	70 L.
R.eq.	2.5 L.	3.9 L.	2.5 L.	1.5-3.5 L.
M.R.M.V. / V.C.	21	22	21	20
C.U.S.	67 per cent	65 per cent	61 per cent	70 per cent
Res.			23 per cent	24 per cent
F.res.det.			45 per cent	40 per cent

1e op. 14.12.51 Ap. post. segm. R. upper lobe
2e op. 16. 4.52 Ap. post. segm. L. upper lobe

On account of pleural adhesions on both sides it was impossible to institute intrapleural pneumothorax, and because the pulmonary function tests yielded satisfactory results it was decided to carry out bilateral resection therapy (*Table* IX & X).

On December 14, 1951, the right apicoposterior segment was removed. A cavity the size of a cherrystone with tuberculous granulation tissue in the surrounding area was found in the operation specimen.

The post-operative course was uneventful and the lung expanded excellently; however, a slight diaphragmatic adhesion persisted.

The functional loss occurring after this operation was negligible, while there was practically no change in the relationship of the work of both lungs (*Table* IX & X).

The second operation was done on April 16, 1952 when the left apicoposterior segment was resected.

The operation specimen yielded a cavity the size of a hazelnut with a considerable amount of tuberculous granulation tissue in the surrounding area. The post-operative course was uneventful, the lung expanded nicely and he is at present in excellent condition.

The pulmonary function may be called good six months after the second operation (*Table* IX & X). The vital capacity and M.R.M.V. have not decreased, but the C.U.S. value has remained on the low side. We cannot give a satisfactory explanation for this. It is possible that the diaphragmatic adhesions are the cause, and that further improvement may occur when the mobilization has lasted longer.

Bronchospirometric investigation showed that the two lungs shared fairly normally in the ventilation and the oxygen uptake, and that, no changes resulted from the second operation.

This case can therefore also be considered as a satisfactory clinical cure with good preservation of lung function.

TABLE X
BRONCHOSPIROMETRY

Date	27.11.51		26.3.52		17.10.52	
	R.	L.	R.	L.	R.	L.
V.C.%	55	45	49	51	52	48
R.M.V.%	56	44	53	47	51	49
O_2%	52	48	54	46	57	43
R.eq.L.	3.2	2.7	2.9	3.1	2	2.6

1e op. 14.12.51 Ap. post. segm. R. upp. lobe
2e op. 16. 4.52 Ap. post. segm. L. upp. lobe

Patient F, a male, age 23, had been ill for a considerable time before operation was decided upon. In July, 1949, he developed pleurisy on the left side, for which he was treated at home. The pleurisy cleared up, but rather extensive changes occurred in the lung; he was admitted to the sanatorium in April, 1950.

The left upper lobe contained an extensive caseating process with liquefaction. In the lower part of the apical segment of the right upper lobe there were some small caseous foci.

The process improved satisfactorily under the influence of bed rest and drug treatment, the sputum became negative. However, a small cavity persisted on the left side, so intrapleural pneumothorax was attempted in August 1950. This was not satisfactory on account of adhesions. The cavity disappeared, however, and he was mobilized to some degree. On the left there remained a number of caseous foci in the apicoposterior segment, but, in June 1951, a tuberculoma the size of a plum developed in the apical segment of the right upper lobe, at the site of the old small caseous foci. Because the pulmonary function was amply sufficient, bilateral resection was considered (*Table* XI & XII).

TABLE XI
SPIROGRAPHY

Date	23.11.51	11.3.52	29.9.52	Normal
V.C.	4180 c.c.	3815 c.c.	3365 c.c	3600 c.c
M.R.M.V.	95 L.	104 L.	84 L.	72 L.
R.eq.	2.8 L.	3.6 L.	2 L.	1.5-3.5 L.
M.R.M.V. / V.C.	22.8	27.2	25	20
C.U.S.	76 per cent	78 per cent	85 per cent	70 per cent
Res.		16 per cent	11 per cent	20 per cent
F.res.det.		35 per cent	31 per cent	40 per cent

1e op. 17.12.51 Ap. segm. R. upp. lobe (subsegm).
2e op. 7. 4.42 L. upper lobe

TABLE XII
BRONCHOSPIROMETRY

Date	23.11.51		11.3.52		29.9.52	
	R.	L.	R.	L.	R.	L.
V.C.%	58	42	55	45	59	41
R.M.V.%	53	47	55	45	50	50
O_2%	60	40	56	44	64	36
R.eq.L.	1.8	2.4	1.8	2	2.3	4.4

1e op. 17.12.51 Ap. segm. R. upp. lobe (subsegm).
2e op. 7. 4.52 L. upper lobe

The processes on the right side were the most circumscribed and this side was operated upon first. On December 17, 1951, the apical segment was partially removed.

A caseous focus the size of a chestnut with a border of granulation tissue was found in the specimen.

The lung expanded excellently, and there was no loss of function (*Table* XI & XII). Because the abnormalities on the left were probably localized in the apicoposterior segment, it was decided to remove this also.

However, during the operation (April 7, 1952) a complication arose, so the whole upper lobe including the lingula was resected.

The specimen showed a caseous focus the size of a chestnut, some smaller foci and areas of bronchiectasis.

The course was uneventful after the second operation and the expansion of the lung was excellent.

Even though in this case more lung tissue was removed than was originally intended, we may be satisfied with the restoration of function (*Table* XI & XII). The vital capacity fell 800 ml. after the two operations, a value approximately corresponding with the loss of an upper lobe. The same is true for the decrease of the M.R.M.V. after the second operation. There was no increase of the residual volume, which accentuates the satisfactory expansion of the lower lobe without overstretching.

Bronchospirometric examination proved that the loss of oxygen uptake in the left lung was only slight compared with the original condition. The oxygen uptake of the left lung had already decreased prior to the first operation, due to the change in the lung tissue caused by the rather extensive process initially present in the apicoposterior segment. We obtained a good result in this case, clinically as well as with respect to the pulmonary function.

SUMMARY

Bilateral resection therapy has yielded surprisingly good results in the first six patients. These were all young people who, even though their tuberculosis was only slightly active, were socially incapacitated owing to their expectoration of weekly positive sputum.

It is of course impossible to say with absolute certainty that they are permanently cured, but this is true for every recovered tuberculous patient.

The most important tuberculous foci have been removed, and it is to be expected that, with prolonged aftertreatment and a sensible way of living, they have a reasonable chance of gaining the fight against the bacillus with their own defensive powers.

It was of course of great importance that the resection was always as selective as possible, and that, apart from the last case, it was always possible to carry out segmental resection.

In our opinion this explains why the loss of function has remained so slight. The total loss of vital capacity and of M.R.M.V. after successful lobectomy is sometimes not much greater than after segmental resection, because it is possible that after the latter operation the spared parts of the lobe participate little in the ventilation and gas exchange (Hirdes). We are of the opinion, however, that when the expansion of the lung is good, these parts are still of significance and that the loss of oxygen uptake of the operated lung can be small (Kraan and Van Der Drift).[5, 6]

The determinations in our patients Nos. 3, 4 and 5 show how slight the loss can be after uncomplicated bilateral resections (*Table* XIII).

TABLE XIII
PULMONARY FUNCTION AFTER BILATERAL RESECTION

	Vit Cap.	M.R.M.V.
Pat. 3	—1.4 per cent	—3.1 per cent
Pat. 4	—3.6 per cent	+9.1 per cent
Pat. 5	—3.6 per cent	—3.6 per cent

We therefore believe that in patients with a localized bilateral affection resection therapy constitutes the only possibility of obtaining clinical cure with complete restoration of fitness for work.

RESUMEN

El tratamiento por resección bilateral, ha dado sorprendentes buenos resultados en los primeros seis enfermos. Estos eran jóvenes que aunque su tuberculosis era sólo ligeramente activa, estaban incapacitados socialmente debido a su expectoración de esputos ligeramente positivos.

Desde luego que es imposible decir con completa certeza que están permanentemente curados, pero esto puede decirse de todo enfermo que se recupera de tuberculosis.

Los focos tuberculosos más importantes, fueron resecados y se espera que con prolongado tratamiento ulterior y manera prudente de vivir, tengan probabilidades razonables de ganar la lucha contra el bacilo con sus propios medios.

Fué de gran importancia por supuesto, que la resección siempre fué tan selectiva como posible y que fuera del último caso, siempre fué practicable la resección segmentaria.

En nuestra opinion, esto explica por qué la pérdida de función ha sido siempre ligera. La total pérdida de capacidad vital y de M.R.M.V. después de una lobectomía satisfactoria no es a veces mucho más grande que después de segmento-resección, porque es posible que después de la última operación mencionada, las partes que quedan del lóbulo participen poco en la ventilación y le cambio de gases. Somos de opinión sin embargo, que cuando la expansión del pulmón es buena, estas partes aún son de significación y que la pérdida de aprovechamiento de oxígeno del pulmón operado, puede ser pequeña. (Kraan y Van Der Drift).

Las determinaciones en nuestros enfermos Núms. 3, 4 y 5, muestran que tan pequeña puede ser la pérdida después de resección bilateral sin complicaciones. Cuadro XIII. Por tanto, nosotros creemos que en enfermos con afección bilateral localizada, la resección constituye la única posibilidad de obtener la curación clínica con restauración de capacidad de trabajo.

RESUME

Le traitement par exérèse localisée portant sur les deux poumons a eu des résultats surprenants chez les six premiers malades opérés. Il s'agissait dans tous les cas de malades jeunes, qui bien que leur tuberculose n'était que faiblement active, taient socialement incapables de toute activité, étant donné l'existence d'une expectoration bacillifère.

Il est bien entendu, impossible d'affirmer que ces malades sont guéris d'une façon définitive, mais il en est ainsi pour tout tuberculeux apparemment stabilisé.

Les foyers les plus importants ont été supprimés, et on peut espérer qu'après un traitement post-opératoire prolongé et un mode de vie convenable, ils ont toute chance de triompher par leur propre défense dans la lutte contre le bacille.

Il est naturellement capital d'insister sur le fait que l'exérèse fut toujours aussi limitée que possible. L'auteur pense qu'ainsi s'explique la modestie du déficit fonctionnel.

Ce déficit fonctionnel n'est parfois pas plus grand après une lobectomie bien réussie qu'après une résection segmentaire parce qu'il est possible qu'après la résection segmentaire, la partie restante du lobe ne prenne plus qu'une part minime dans la ventilation et les échanges gazeux. Néanmoins, l'auteur pense que lorsque le poumon subit une bonne expansion, les parties restantes du lobe gardent une valeur certaine et que la perte du pouvoir d'oxygénation du poumon opéré peut être légère.

Chez les malades 3, 4 et 5, on voit combien la perte peut être légère des résections bilatérales sans complications. C'est pourquoi l'auteur pense que chez des malades atteints de lésions bilatérales localisées, l'exérèse réalise la seule possibilité d'obtenir une reprise d'activité professionnelle.

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Analysis of 77 Consecutive Cases Receiving Pneumoperitoneum, Rest and Chemotherapy*

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Introduction

When chemotherapy is added to bed rest, a much greater number of tuberculous cavities close. The following data indicate that when lung volume is reduced by pneumoperitoneum a further number of cavities close. If pneumoperitoneum is then continued for several years, the relapse rate is low.

A follow-up and analysis were recently completed on 77 consecutive cases of pulmonary tuberculosis receiving routine sanatorium care, chemotherapy and pneumoperitoneum in Gaylord Farm Sanatorium between December 31, 1948 and December 1, 1952. The average census in this semi-private sanatorium was 120. Previously published reports of pneumoperitoneum therapy before chemotherapy coverage showed a high percentage of failures and a mortality of from 5 to 30 per cent. There has been no tuberculosis mortality in this series. Excluded from the series was a patient who went home against medical advice after 73 days of pneumoperitoneum treatment.

The 77 consisted of 61 per cent far advanced, 35 per cent moderately advanced and 4 per cent progressive minimal instances of tuberculosis. All had positive sputum before pneumoperitoneum treatment. Bilateral disease was present in 83 per cent. Cavitation was present in 71.4 per cent. There were 20.8 per cent with multiple cavities, and 18.2 per cent with bilateral cavities. Predominantly exudative and fresh lesions made up 44 per cent, fibrotic lesions 27.3 per cent and mixed lesions 28.7 per cent. Men were in the slight majority at 56 per cent. Medium age for men was in the late 30's and for women in the late 20's. Table I shows the number

TABLE I
AGE OF ALL PATIENTS

Age Group	16-25 Years	26-35 Years	36-45 Years	46-55 Years	56-61 Years	Total No.	Total Per Cent
Men	8	12	10	10	3	43	56
Women	11	16	5	2	0	34	44
Total	19	28	15	12	3	77	

of men and women in each group. These 77 patients as a group had more disease and a worse initial prognosis than the rest of the sanatorium patients. Of the 77 cases 23 had either previous or attempted pneumothorax. Three had previous thoracoplasties.

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Most cases received bed rest and chemotherapy on entering the sanatorium, and pneumoperitoneum was often not added until a reasonable doubt existed that cavity closure would take place without it. Many were given pneumoperitoneum with eventual surgery in mind. General clearing and cavity closure were common and in those cases surgery was not further considered.

Results

Of the 77 cases 29 have had pneumoperitoneum terminated and an additional 13 have had surgery. In December 1953 there were 35 continuing pneumoperitoneum. The number returning to society to lead useful lives totalled 71 out of 77. An additional two cases had retired from work because of age. Another was physically well but had mental illness. One is recovering from successful surgery. Two remain with pneumoperitoneum and open cavity. One left against medical advice and improved after refusing surgery. He has negative sputum and has returned to work after prolonged chemotherapy. The other is the only case remaining with positive sputum out of 77. This patient had thoracoplasty and was given pneumoperitoneum to control contralateral spread with cavity. The condition is improved but the cavity is still open. The one death was unrelated to tuberculosis and followed a coronary attack 20 months after thoracoplasty had been substituted for pneumoperitoneum, and nine months after return to work.

All 22 without cavities showed marked retrogression of lesions by serial roentgenograms. Sputum disappeared and routine gastric lavage became negative on culture and by guinea pig inoculation. Some of this group had a very marked degree of bilateral infiltration before treatment.

Cavity Closure Principal Target

The most important single target was cavity closure. This aim was achieved by bed rest, chemotherapy and pneumoperitoneum in 76.7 per cent of cavities and by abandoning pneumoperitoneum for operation in a further 20.5 per cent. It is necessary to distinguish between the number of cavities and the number of patients with cavities. Those with cavities

TABLE II

DISTRIBUTION OF 73 CAVITIES

Lobes	No.	%	No.	No.	No.	%	No.	%	No.	%	No.	%	No.	%
	R.U.		R.M.	R.L.	Rt. Side		L.U.		L.L.		L. Side		Total	
Number of Cavities	38		0	2	40		27		6		33		73	
Closed by Pneumoperitoneum	32	84.2	0	2	34	85	17	63	5	83.3	22	66.7	56	76.7
Closed by Operation	5	13.2	0	0	5	12.5	9	33.3	1	16.7	10	30.3	15	20.5
Total Closed	37	97.4	0	2	39	97.5	26	96.2	6	100	32	97	71	97.2
Cavities Uncollapsed	1	2.6	0	0	1	2.5	1	3.8	0		1	3	2	2.8
Total	38	100	0	2	40	100	27	100	6	100	33	100	73	100

closing with pneumoperitoneum were 72.7 per cent and with operation 23.6 per cent, a total of 96.3 per cent. Table II shows the distribution of the 73 cavities in the series, and the number closing with pneumoperitoneum or operation. It is seen that a high percentage of cavities collapse readily in any lobe. New cavities collapse quickest. A definite relationship was found to exist between the length of time from sanatorium entrance to pneumoperitoneum induction and the length of time needed for cavity closure after pneumoperitoneum. The longer pneumoperitoneum induction was postponed the longer it took to close excavation. Table III shows that the

TABLE III

AVERAGE TIME REQUIRED FOR CAVITY CLOSURE WITH PNEUMOPERITONEUM

Type of Lesion	Number of Patients	Number of Cavities	Average Sanatorium Residence before Pneumoperitoneum	Average Time Required for Closure with Pneumoperitoneum	Total Sanatorium Residence before Closure
Exudative	18	27	2 months	2 months	4 months
Mixed	12	15	9.2 months	4 months	13.2 months
Fibrotic	11	14	7 months	7.6 months	14.6 months

average time taken for closure of the 27 cavities in 18 exudative lesions was two months. The average time for closure of 15 cavities in 12 mixed lesions was four months, and the average time of the 14 cavities in 11 fibrotic lesions was 7.6 months. There was a great difference in the length of sanatorium care before pneumoperitoneum induction between the exudative cases and the other two groups. The average sanatorium stay before pneumoperitoneum in the exudative cases was two months, in the mixed cases 9.2 months, and in the fibrotic cases 7 months. Many of the mixed and fibrotic cases had previous sanatorium admissions to account for the long average sanatorium residence before pneumoperitoneum. The mixed cases had predominantly old disease with recent exudative spreads. When the average length of sanatorium care before pneumoperitoneum was added to the average time taken for cavity closure after pneumoperitoneum, the exudative cases had their cavities closed in four months, the mixed in 13.2 months, and the fibrotic in 14.6 months.

TABLE IV

THE 18 EXUDATIVE CASES IN TABLE 3 DIVIDED INTO THOSE RECEIVING PROMPT PNEUMOPERITONEUM INDUCTION AND THOSE IN WHOM INDUCTION WAS DELAYED

Type of Lesion	Number Patients	Number Cavities	Average Sanatorium Residence before Pneumoperitoneum	Average Time Required for Closure with Pneumoperitoneum	Total Sanatorium Residence before Closure
Exudative (Delayed Pneumoperitoneum Induction)	8	9	4 months	2.4 months	6.4 months
Exudative (Prompt Pneumoperitoneum Induction)	10	18	½ month	1.6 months	2.1 months

In the 22 exudative lesions with cavities, the longer pneumoperitoneum induction was delayed the longer it took to close the cavities after pneumoperitoneum was given (Table IV). There were 10 patients with 11 cavities who had pneumoperitoneum induction averaging four months after admission. One with a single cavity was operated on and another refused operation. The other eight with nine cavities obtained cavity closure 2.4 months after pneumoperitoneum induction, or 6.4 months after sanatorium admission. The remaining 12 with exudative lesions had 20 cavities. Two of them with single cavities were operated. The remaining 10 had 18 cavities. This sub-group was the worst in the whole series and pneumoperitoneum induction was not delayed. It was started in the first month, two weeks being the average time. These 18 cavities closed with pneumoperitoneum in an average of 1.6 months, or 2.1 months after admission. Table V shows the size of these 18 cavities. Because of these results, prompt

TABLE V

SIZE OF THE 18 CAVITIES IN 10 EXUDATIVE CASES RECEIVING PROMPT PNEUMOPERITONEUM INDUCTION FOLLOWED BY RAPID CAVITY CLOSURE

Size of Cavity	1.5 cm. to 2.4 cm.	2.5 cm. to 3.4 cm.	3.5 cm. to 4.4 cm.	4.5 cm. to 5.1 cm.	
Number of Cavities	7	5	4	2	18 Total

pneumoperitoneum induction is believed to be justified, not only because fresh cavities collapse quickest and delay may prevent collapse, but also because delay encourages spread of disease and resistant organisms. Small recent cavities should be given several weeks for possible spontaneous closure before pneumoperitoneum consideration. One of the reasons given to explain most failures of prolonged streptomycin-para-aminosalicylic acid rest treatment of tuberculosis in a series of 250 patients at Trudeau Sanatorium,[1] whose treatment was begun between August 1949 and June 1952, was failure to use pneumoperitoneum from the outset in the presence of cavity of more than 1 cm. in diameter.

Surgery

Table VI shows the type of surgery used on the 13 patients operated following pneumoperitoneum, as well as the type of disease present. Tho-

TABLE VI

SURGERY

NUMBER IN EACH TYPE RECEIVING SURGERY, AND TYPE OF SURGERY WITH RESULTS

Type	Number of Cases	Resection	Resection and Thoracoplasty	Thoracoplasty	Result
Exudative	3	1	1	1	Good
Mixed	6	2	1	3	Good
Fibrotic	4	2	1	1	Good

racoplasty was the operation of choice up to 1950. Since then it has been replaced by resection with or without thoracoplasty.

The majority of articles written on pneumoperitoneum have mentioned temporary phrenic paralysis as a usual accompaniment. Phrenic crush was used in six cases in this series and there were three successes. It is believed that this operation is unnecessary in the great majority of cases. It is known that the operation is not without danger since it may cause permanent partial or occasionally permanent complete paralysis of the diaphragm.[2] In chest surgery today emphasis is placed on the importance of conserving as much normal lung function as possible. Restricting the use of phrenic crush in pneumoperitoneum therapy is one way of furthering this aim.

Chemotherapy and Relapses

In the chemotherapy used in this series there were great variations in length of treatment and dosage in different combinations of para-aminosalicylic acid, streptomycin, dihydrostreptomycin and isoniazid. The most common combination was para-aminosalicylic acid and streptomycin. A large proportion of the earlier cases received inadequate courses according to 1953 standards. The short term chemotherapy cases have now been followed from four to five years compared to from one to three years for the long term cases. Twenty-one received one to three months of chemotherapy; 12 four to seven months; 16 eight to 11 months and 28 twelve months or more. Three of the five relapses were in the group receiving one to three months of drugs, the fourth case had four months and the fifth case one year. There were two serious relapses during pneumoperitoneum therapy with cavity reappearing after sanatorium discharge. One was admitted to another sanatorium and had successful resection. The other was re-admitted for further chemotherapy and rest. The cavity again closed and pneumoperitoneum has now been terminated. The remaining three relapses consisted of new or reactivated infiltrates measuring from 1 cm. to 3 cm. and with negative sputum. The relapse rate to date is thus 6.5 per cent compared to a readmission rate of 22 per cent for the sanatorium. Extent of disease for all sanatorium cases from 1949 to 1952 was far advanced 14.8 per cent and this pneumoperitoneum series 61 per cent, moderately advanced 29.8 per cent and this series 35 per cent, minimal 29.8 per cent and this series 4 per cent. Little or no chemotherapy for minimal and moderately advanced cases on first admission is believed to have been a major factor in the higher general readmission rate. All pneumoperitoneum cases not only received chemotherapy, but they had special attention throughout the period of pneumoperitoneum maintenance such as frequent x-ray films, flouroscopic examinations, weight checks and general interrogations. With the now general use here of prolonged chemotherapy for all stages of the disease one would anticipate a drop in the general readmission rate within a few years.

Complications

Complications were few and they did not necessitate stopping pneumoperitoneum. There was one small sterile peritoneal effusion. Surgical emphysema developed on several occasions. Three diabetics and three with inguinal hernia responded to pneumoperitoneum. One had a urinary stone during pneumoperitoneum.

Technique

There is agreement with Banyai and Trimble[3] that a two week interval for pneumoperitoneum refills is too long. The favored schedule is a weekly one, and a 10 day interval is occasionally used. The desired size of the pneumoperitoneum can usually be obtained in a week. It is then important to find the proper amount of air necessary for weekly maintenance. It usually varies between 600 cc. and 1000 cc., depending upon the size of the patient.

Length of Treatment

In the cases terminated to date without surgery, the duration of pneumoperitoneum has been 28 months, with the first 11 months having been in the sanatorium. Fifteen were women and three of them have since successfully completed pregnancy. In pneumothorax it was usually considered advisable to maintain collapse for several years in order to try to prevent relapses and cavities from re-opening at the end of the treatment.[5] The same policy was followed in this series. When the stay in the sanatorium has ended and prolonged chemotherapy has been completed, pneumoperitoneum then being continued alone may be considered as an air cushion for the lungs in the trying months ahead. Since good health is the rule in this period it is reasonable to believe that development of resistance to the disease will then be encouraged. Amberson[6] has often stressed the importance of resistance development for longevity.

SUMMARY

Seventy-seven cases of pulmonary tuberculosis receiving chemotherapy, rest and pneumoperitoneum at Gaylord Farm Sanatorium between December 31, 1948 and December 1, 1952 are presented of whom 61 per cent were far advanced, and 71.4 per cent had cavitation. All had positive sputum before treatment and one remained positive. Thirty-five continue to receive this treatment.

Cavity closure was achieved with pneumoperitoneum in 72.7 per cent of cavity cases with surgery in a further 23.6 per cent. Fresh cavities closed quickest. Average time for closure of 27 cavities in 18 exudative cases was two months, 15 cavities in 12 mixed cases four months, and 14 cavities in 11 fibrotic lesions 7.6 months.

The longer pneumoperitoneum induction was postponed the longer it took to close excavation. Average sanatorium stay before pneumoperitoneum induction added to time taken for closure after induction showed exudative cases closed in four months, mixed in 13.2 months and fibrotic in 14.6 months.

Subdividing the 18 exudative cases having 27 cavities, eight had nine cavities with an average pneumoperitoneum induction delay of four months. Closure took another 2.4 months for a total of 6.4 months after admission. The remaining 10 patients had 18 cavities with pneumoperitoneum induction in the first month, two weeks being the average time. Closure took another 1.6 months for a total of 2.1 months after admission. This latter sub-group was the most acute in the whole series.

Complications were few and unimportant. Of the five relapses, two were serious but they recovered. Four of the relapses occurred in patients who had had short courses of chemotherapy.

Prompt pneumoperitoneum induction is believed to be justified, not only because fresh cavities collapse quickest and delay may prevent collapse, but also because delay encourages spread of disease and resistant organisms.

SUMARIO

Se presentan 77 casos de tuberculosis pulmonar tratados con quimioterapia, reposo y neumoperitoneo en el "Gaylord Farm Sanatorium" entre Diciembre 31 de 1948 y Diciembre 1° de 1952; de ellos, 61% eran casos muy avanzados y 71.4% tenían caverna. Todos tenían esputo positivo antes del tratamiento y uno permaneció positivo. Treinta y cinco continúan recibiendo este tratamiento.

Se obtuvo el cierre de la caverna con neumoperitoneo en 72.7% de los casos cavitarios, y, con cirugía en un 23.6% más. Las cavernas jóvenes cerraron más rápidamente. El tiempo promedio de cierre de 27 cavernas en 18 casos exudativos, fué de dos meses; cuatro meses en 15 cavernas de 12 casos mixtos; y 7.6 meses en 14 cavernas de 11 lesiones fibróticas.

A mayor tiempo de retardo en la inducción del neumoperitoneo, mayor tiempo se necesitó para el cierre de la caverna. El promedio de estancia sanatorial previamente a la inducción del neumoperitoneo, agregado del tiempo necesario para cerrar la lesión después de inducido, mostró ser en los casos exudativos de 4 meses, en los mixtos de 13.2 meses y en los fibróticos de 14.6 meses.

Subdividiendo los 18 casos exudativos que tenían 27 cavernas, 8—tuvieron 9 cavernas con un promeido de retardo en la inducción del neumoperitoneo de 4 meses. El cierre tomó otros 2.4 meses o sea un total de 6.4 meses después de la admisión. Los restantes 10 pacientes tuvieron 18 cavidades con inducción de neumoperitoneo en el primer mes, siendo el promeido de tiempo de dos semanas. El cierre tomó otros 1.6 meses, para un total de 2.1 meses después de la admisión. Este último sub-grupo fué el más agudo de todas las series.

Las complicaciones fueron pocas y sin importancia. De las cinco recaídas, dos fueron serias, pero se recuperaron. Cuatro de las recaídas acontecieron en pacientes que habían tenido cursos cortos de quimioterapia.

RESUMÉ

L'auteur présente 77 cas de tuberculose pulmonaire traités entre le 31 décembre 1948 et le Ier décembre 1952, par la chimiothérapie, le repos et le pneumopéritoine, au Sanatorium Gaylord Farm. 61% d'entre eux

étaient des cas très graves et 71,4% étaient atteints de cavernes. Tous avaient une expectoration positive avant le traitement et l'un l'est encore resté. 35 malades continuent à être traités de la sorte.

La fermeture des cavernes fut obtenue grâce au pneumopéritoine dans 72,7% des cas et grâce à la chirurgie dans 23,6%. Les cavités récentes disparurent plus rapidement. Le temps moyen de fermeture des 27 cavités dans 18 cas de tuberculose ulcéro-caséeuse, fut de deux mois. Il fut de quatre mois pour 15 cavernes, dans 12 cas de lésions diverses et de 7,6 mois pour 14 cavités appartenant à onze cas de lésions fibreuses.

Plus le pneumopéritoine tarda à être institué, plus les cavernes mirent de temps à se fermer. La moyenne de séjour en sanatorium avant l'établissement du pneumopéritoine, aditionné au temps de la fermeture de la cavité après mise en oeuvre de ce traitement donne:

—tuberculose ulcéro-caséeuse, fermeture en 4 mois.

—lésions diverses, fermeture en 13 mois, 2.

—formes fibreuses, fermeture en 14 mois, 6.

Si on analyse les 18 case de tuberculose ulcéro-caséeuse pour lesquels il existaient 27 cavités, dans 8 cas il y avait 9 cavités pour lesquel les on attendit une moyenne de 4 mois avant la création du pneumopéritoine. Il fallut 2 mois, 4 supplémentaires pour amener la fermeture de ces cavités, soit au total 6 mois, 4 après l'admission. Les dix autres malades pour lesquels il y avait dix cavernes furent soumis au pneumopéritoine dans le premier mois (en moyenne au bout de deux semaines). La fermeture des cavités nécessita 1 mois, 6 en principe, soit au total 2 mois, 1 après leur admission. Ce dernier groupe est celui dont l'évolution fut la plus aiguë.

Les complications furent rares, et sans gravité. Parmi les cinq rechutes, deux furent sérieuses, mais la guérison survint néanmoins. Quarte des rechutes survinrent chez des malades qui avaient été traités par le chimiothérapie pendant un court laps de temps.

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Diagnosis of Hydatic Pulmonary Cyst

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Radiologic Diagnosis of Hydatid Pulmonary Cyst

When x-rays were discovered in 1895 specialists started to describe the radiologic aspects of the pulmonary diseases. Levy-Dorn and Zadek affirmed that the characteristic picture of an entire simple cyst in the lungs is a round shadow with neat borders. As every symbol in biology, this affirmation proved to be false and so Lanari was able to say that the most perfect image of an hydatic cyst he had seen corresponded to a sarcoma.

The studies continued and Claessen gave an important step in his treatise of hydatidosis. Later the works of Sergent, Beclere, Tuffier, Nemenow, Escudero, Deve, Belot, Lozano, Morquio, Petzetakis, Peuteuil, Stephani, Guarini, Liemann, Lagos Garcia, Queirolo and others made the problem of the diagnosis of hydatic cyst reach its full maturity. For a better study we shall make an anatomoradiologic division and thus find:

A). *Unbroken cysts:* The density of the shadows on the roentgen picture is proportioned to the specific weight of the bodies that appear in it—since the specific weight of the whole cyst—its mother-bag, its two walls and the liquid it contains is greater than that of the surrounding air, hydatic cyst can be easily seen in the lungs. Tuffier asserted that x-rays can pass through the hydatic cyst without producing a shadow. Recently, Escardo and Anaya stated that an hydatic cyst smaller than an egg cannot be seen on the x-ray screen. We find both affirmations false, for reasons explained above and because we have seen simple, not calcified cysts as small as a cherry produce a picture and the vomica or the operation proved they were hydatic cysts.

The difficulty of diagnosis consists in differentiating hydatic cysts from other bland parts of the same density, like tumors, inflammations, parasites etc. The resemblance of their shadows with those of hydatic cysts is the reason there are no pathognomonic images in hydatidosis of the lungs but only characteristic ones.

The roentgenologic image of an unbroken simple cyst depends greatly upon its localization. Central cysts generally develop according to the law of Belot and Peuteuil which reads: Every neoformation in the lungs tends to adopt a spherical form because the density of the lung tissues is small and homogenous. There is no axis of expansion, for the forces of peripheral attraction of the lungs are equally distributed and influenced only by the changes of intrathoracic pressure due to respiration. In consequence, Beclere asserts that every hydatic cyst produces a round, dark, frequently spheric, homogenous shadow with neat borders, as if drawn with a compass, easily seen against the clarity of the surrounding parenchyma. This concept is widely accepted and supported by Holsknecht, Didie, Picort, Auspensky, Morquio etc., while others criticize it, particularly Escudero

who affirms that the most exactly round pictures are produced by pulmonary sarcoma.

There are many authors between these two criteria, including ourselves; we find that the radiologic image of an entire simple cyst can vary from a perfectly round shadow (Fig. 1) to the most strange shapes like the one represented by the Fig. 2. We consider that the factor of utmost importance in the form of the cyst is its localization, because all the cysts situated near the thoracic wall, diaphragm or mediastinum are deformed by the rigid parts that limit their growth. The entire cyst presents a dense shadow with rather well determined borders. One of the most characteristic signs of an entire hydatid cyst is that of separation: in the upper part of the cyst, between the adventitia and the germinative capes, there is a clear space in form of a half moon with the concavity downwards. This clear space is the pneumoperivesicle, the so called "calotte aerienne" of Deve. Another rather frequent sign is that of the buoy—it is a shadow in the interior of the parenchyma, seemingly floating. It is easily influenced by respiration (sign of Escudero-Nemenow). During inspiration it adopts a vertical form, during expiration the contrary occurs. In our investigation we have seen this sign in 45 per cent of the cases; the Valsalva maneuver makes this sign even more accentuated. The further from the hilus are the cysts, the more they move with the respiration, except when they have strong adherences or are caught between two bronchial tubes. When a cyst gets caught between two bronchial tubes it also gets deformed while growing and so a cyst which was completely circular when small, may become polygonal after a certain time. This is very rare, it is much more frequent to find such polygonal forms in the emptied hydatic cysts because of the loss of the intracystic tension.

In unbroken cysts the Queirolo-Walsch statement is of a great diagnostic value. It says: while pulmonary cysts reject the bronchial tubes, tumors, on the contrary attract and deform the aerial ways and later even the thoracic walls and the mediastinum. Mansoury thinks that hydatid cyst permits the outline of the lung to be seen in its shadow, but that

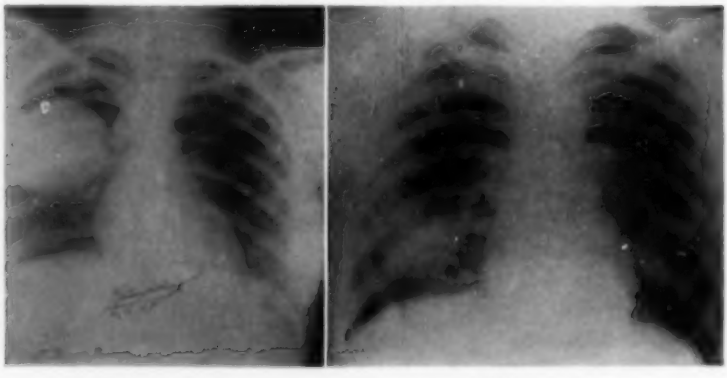

FIGURE 1

FIGURE 2

tumors do not. Spender, Viallet and other authors do not give this proposition any value because the contour of the lungs can be seen in almost all the dense shadows of the thorax; it depends on the technic employed and the volume and situation of the shadow. The visibility or invisibility of the lungs serves us only to determine if the process affects the whole lung. It is in the saclike aneurysm that we see least of the pulmonary tissues.

Hydatid pulmonary cysts frequently present shadows with marked protuberances; the borders in these cases may still be neat and precise, or, they may have an irregular, dentiform and oily outline that Escudero and other Argentine authors have often found. Some authors (Broc, Jaubert de Beaujeau, Remy-Roux and Aubry) assert the reason for these oily limits is that in certain cases the bags of the new generation grow through the cuticle toward the outside instead of growing inside. This is especially common among animals, but Kilvington found it rather frequently in the human liver, too which makes that localization of the disease so much more serious. Other authors confirmed recently this phenomenon which sometimes originates a radiographic image in form of a potato (Fig. 3) or of a halteridium.

Another serious difficulty encountered in the diagnosis of hydatid cysts consists in determining whether a cyst is located in the lower part of the lungs or in the liver. There are different means of investigating such a case. Lateral radiography will clear doubts in many cases. Blefari recognizes a great value of the liberation of the costophrenic sinus (Fig. 4) which is frequently occupied by the cysts and other subphrenic tumors and usually remains free in case of pulmonary hydatic disease. We find that affirmation correct but would add that another important sign is the liberation of the cardiophrenic sinus in pulmonary hydatid cysts, while subphrenic cysts and tumors tend to occupy it; (particularly diaphragmatic hernia). We can also use to great advantage the diagnostical pneumoperitoneum; the diagnostical pneumothorax advised by Abadie, on the contrary, seems to us of less value and is most of the times impossible to realize because of the strong adherences. To determine whether a cyst

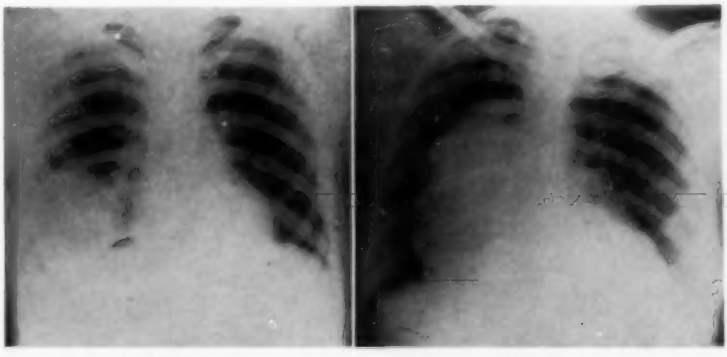

FIGURE 3

FIGURE 4

is located in the liver or in the lower part of the lungs we can also use tomography and radiography with contrast.

Even more difficult to diagnose are cases in which the disease is located in the lung and in the liver at the same time. Frequently it is necessary to proceed to a thoracotomy or a thoracolaparotomy in order to be able to make a diagnosis. Keeping in mind what we have previously said we can distinguish the following anatomoclinical forms of an unbroken cyst:

1) *Isolated cyst, without generative capacity.* Frequently in the lungs there is just one cyst, without vesicles. Sometimes it is quite big and produces few symptoms. The germinal membrane of the cyst can be separated easily, it is smooth and gets thinner as the cyst grows. The external membrane is united with the viscera itself. Spontaneous opening of this cyst is fairly frequent, sometimes giving way to vomica. A real degeneration of the tissues surrounding the cyst caused by the external membrane was observed from time to time. Escudero has studied this condition and has given it the name of cuticular degeneration. Clinically we call these patients silent. The discovery of the disease is very often fortuitous.

2) *Secondary bronchogenic echinococcosis of Deve and Herbert.* This form is developed by a cyst with germinative capacity. After invading the bronchial tree it can, by aspiration, provoke multiple infiltrations in parts far from the primitive cyst. It is often accompanied by acute manifestations in the respiratory ways such as pleuritis, atelectasis and infections of the residual cavity, so that the general symptomatology is usually accentuated.

3) *Primitive and hematogenous multiple pulmonary hydatidosis.* These infiltrations are originated from the intestinal chyliferous reached by the eggs by way of ingestion. The eggs pass by the vena porta to the liver, from there by the suprahepatic veins to the inferior cava, right part of the heart and to pulmonary artery which takes them to the lungs.

4) *Secondary pulmonary hydatidosis, single or multiple.* This form is characteristic of old carrier patients who have had hydatid cysts outside of the thorax. The symptomatology varies greatly. It is extremely frequent that the primitive localization is in the liver. These cysts have a

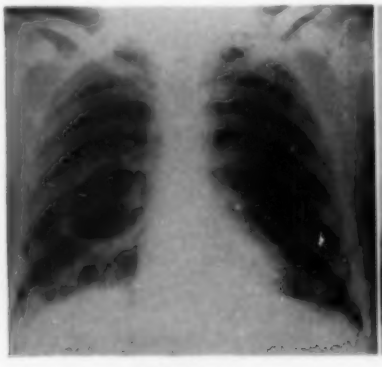

FIGURE 5

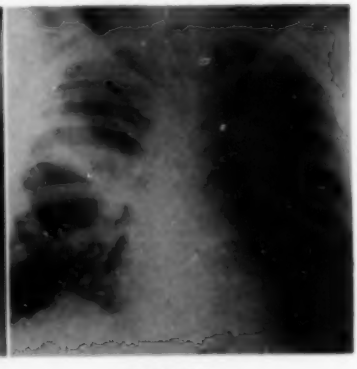

FIGURE 6

tendency to open spontaneously in bronchi or other organs.

Hydatid cysts can also be situated in the pleura. The primitive echinococcosis of the pleura was first described by Gary and Lion in 1900 and confirmed by Deve's experiences in animals. The hexagon-shaped embryo can reach the periphery of the lung by way of blood vessels from where it passes to the pleura and remains there. Dew does not admit this primitive localization, and we have not seen it. We rather think that in the majority of cases pleural cysts have origin in a pulmonary cyst that had enucleated itself and passed to the pleura. When the parasite grows in the pleura it compresses the lung, makes the thoracic wall bulge and displaces the mediastinum. In some cases it is eliminated by vomica, as described by Perazzi. Usually this vomica is preceeded by the formation of a pyopneumothorax. Puncture is dangerous in cases of pleural cysts; it is better to do thoracotomy, having previously prepared for an operation in case the diagnosis is confirmed. Pleural hydatid cysts give a predominantly thoracic symptomatology, thus completely different from that of the pulmonary cyst.

The diagnosis of unbroken hydatid pulmonary cyst should be made by way of anteroposterior radioscopy, lateral and oblique radiography, in some cases.

B). *Aseptic complications of unbroken hydatic cysts:* The unbroken cyst can provoke pneumonitis around it or produce a zone of atelectasis. This can obscure the cyst. The unbroken cyst can also be accompanied by pleural reaction caused by osmosis of the hydatid liquid. Depending on the intensity of this reaction we can or cannot see through the cyst.

Calcification of hydatid cyst is a relatively rare process and the only part of it that calcifies is the fibrous capsule. Hydatid cyst in calcified form is often found during autopsy or in a series of radiologic examinations. It is frequently confused by the radiologists with tuberculous processes in which calcifications are more common.

C). *Septic complications of hydatic pulmonary cyst:* The most frequent complication of hydatid pulmonary cyst is perforation of a bronchus. When this occurs many radiologic signs corresponding to the anatomopathological modifications that this cyst undergoes may be observed; only exceptionally the cyst opens without producing radiologic signs. When the liquid is completely expelled, together with the membrane (the curative vomica) the image is exactly the same as after an intervention with actual methods; we observe on the x-ray screen a residual cavity without much perifocal reaction; the cavity is sometimes filled with blood. When the spontaneous opening of the cyst is recent we observe in some cases a zone of condensation in almost all the affected lobes. This zone of condensation may develop into carnification which sometimes, as we have noted ourselves, is almost impossible to see by the x-ray film. When this reaction is minimal and if the expulsion of the membrane and the liquid is partial, a horizontal superior limit above the aerial camara is observed—that means an hydatic pneumocyst was formed. Lagos Garcia found a picture that he described as the sign of "camalote." A picture of this type

is obtained when the patient moves in front of the radioscopic screen—the remainder of the membrane can be seen floating and moving. The membrane rises above the level of the liquid and comes down again, because of its greater density, when the patient remains still. This never happens when the drainage by the bronchus or the catheter is sufficient (Fig. 5). Ivanissevitch finds in these half emptied cysts a sign of the "double arch" that appears in the initial periods of the emptying. Under the line of the adventitia we see an air layer and under it the level of the liquid, but between this level and the adventitia we can observe another line that is the shadow of the partially collapsed membrane.

Another possibility is the total expulsion of the liquid and the persistence of the membrane. In these cases we observe a circular cavity with an opaque body retained in the declivity. This body moves when the patient changes from the Trendelenburg's to the vertical position, passing from the superior to the inferior pole. This, of course, can only be seen when the opening of the cyst is relatively recent, because, on the contrary, the adherences of the membrane do not permit the cyst to float this freely. When the hydatic residual cavity is old it is extremely similar to a tuberculous cavern, especially to an insufflated one (Fig. 5). The only difference is that the hydatic cavity is much neater and clearer than a tuberculous cavern. It often looks somewhat like a pulmonary abscess, particularly when the cyst is infected.

Hydatid pulmonary cysts of the base which open into the bronchi are sometimes difficult to diagnose because of their resemblance with hepatic cysts that open into the bronchia. The presence of bile and many young vesicles in the expectoration cause one to suspect primitive hepatic localization. A profound radiography and a radiography with a contrast will be helpful to establish the origin of the cyst.

It is characteristic for the cyst opened into the bronchi that immediately an infection appears, because of the destruction of the cuticle which

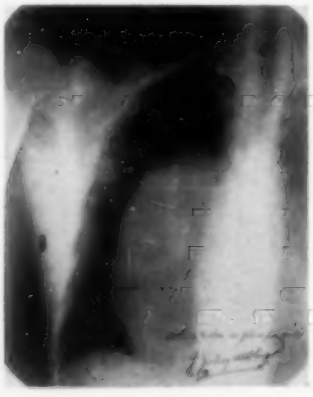

FIGURE 7

The cases quoted are from Spain. The hydatidosis does not exist in Costa Rica.

served as a filter for the germs, always present in the bronchi. Generally the perifocal reaction is not as great as in abscesses and even though the general symptomatology is that of pulmonary infection, the presence of hydatid material in sputum clears any doubts that may arise. In infected cysts one also finds the Escudero sign, not observed in abscesses. The gangliar reaction is also often present in these cases. There is a great group of infected cysts in which the membrane gets loose and separates itself. Tillier observes an irregularity of the superior level of the liquid, the movements of this level being very slow; this sign was described by Cumbo in 1921. It corresponds to the image described by Lozano for the liver and what this author thought was due to the contraction of the young bags. This form may be considered pathognomonic of the infected hydatic pulmonary cyst. Sometimes, says Arce, we find a space full of air between the capsule and the membrane, a sign frequently observed in the beginning of the process of emptying. The superior limit of the cyst can be seen convexed, the air remaining above and visible as a dense shadow in form of an arch. This image, too, is very characteristic of the infected cyst.

Although pulmonary cyst can perforate the pleura, this spontaneous rupture is not frequent particularly because when the cysts grow much they produce fuses of the pleuras. In cases where the pleura has been perforated by hydatid cyst often an hydropneumothorax with its air camara and the liquid level is seen or, even more typical, a hydatid hydropneumothorax that rapidly becomes a pyopneumothorax. Perforation of the pleura by a hydatid cyst gives many symptoms like the asphyxiant valvular pneumothorax, the syndrome of an "acute thorax," abundant vomicas, violent pains, cutting or producing a sensation of an interior tearing. In one of our cases the rupture was produced in a larval way, with no symptomatology.

Cases of secondary pleural echinococcosis are rare: in 1925 Deve quotes as the only ones those of Leonard, Renon and London; later a few others were described by Bernou, Fruchard, Gain and Ugon.

It is much more frequent that the pleura is infiltrated from hepatic cyst which produces what is called colepyopneumothorax. The frequency of this complication seems to be due to the facility with which hydatid cyst of the liver grows upward because of negative intrathoracic pressure; atrophy of the diaphragm by compression (Deve); infection that fuses the diaphragm with the pleura and the necrosis of the adventitia. As far as diagnosis is concerned the presence of bile in the pleural exudate is meaningful. Other signs which help to establish the primitive hepatic localization of the disease are: the sign of the "volcano in eruption" (Fig. 6) described by Harris in which the curved point of the diaphragm indicates the localization of the primitive cyst; the sign of camalote and that of the arcade described by Deve and Levertisseur. This last sign consists in the appearance of a lineal shadow similar to a descending arch which is submerged in the liquid cape; this arch is the dead adventitial bag that remains suspended in the cavity after the separation from the hepatic tissue. The discovery of calcifications in the liver is helpful, for they are

pathognomonic. Perforation of a hepatic cyst in the pleura can also occur in a dissimulated way and without the formation of pneumothorax (the case quoted by Reboul). Spontaneous rupture of a pulmonary hydatid cyst into the abdomen is not known in the medical literature.

Subphrenic hydatid cyst, as Rendu and Deve observe, can break directly in the bronchia through a short trajectory without cavity in the lungs. In this case typical vomica containing membranes and small vesicles takes place; the radiologic image is characteristic. There may or may not be an intermediate pulmonary cavity, but the formation of a basal cyst-like empyema is constant. This empyema can begin by a simple trajectory and due to the continuous passing of the infected material the pulmonary parenchyma gets destroyed; this is followed by a formation of a focus of suppuration. Claessen admits the perforation in the incisura interlobaris; from there it can reach the hilus producing only few disturbances in the lung. It occurs very seldom, due, Deve believes, to the fact that the pleural leaves of the incisura become fused, thus making the penetration impossible. Piaggio Blanco and Garcia Capurro were able to quote 20 cases proving the contrary and they affirm that "the best radiologic images of the liquid interlobar process correspond to pathologic processes originated by subphrenic hydatidosis. Sometimes we find an interlobar pleural reaction in form of incisuritis."

Less frequent, or rather exceptional complications are rupture of the cyst in the pericardium causing pericarditis which begins more or less abruptly. The cyst can also open in the rachidial conduct, skin or the digestive apparatus. Tuberculosis associated with pulmonary hydatidosis gives no characteristic radiologic image. The diagnosis must rely on examination of sputum.

Operated cysts can present other complications one of them being the formation of residual fistulas. These are now rare. Diagnosis of fistulas is made primarily by way of radiographies with a contrast, tomography and the test of methylene blue.

Diagnosis of pulmonary sclerosis is difficult, in fact it sometimes cannot be made except during surgical intervention. Other times radiography is useful; if the sclerosis is accompanied by bronchiectasis the injection of lipiodol in the bronchi is necessary in order to make a diagnosis.

The diagnostic value of tomography in pulmonary hydatidosis is not great, but it is useful in the differential diagnosis, to localize the cyst and in some special cases like the one in Figure 7. Bronchoscopy is helpful in some cases, especially when atelectasis is present.

SUMMARY

The order of the radiologic exploration of pulmonary hydatid disease should be as follows: 1) simple radiography; 2) posteroanterior or anteroposterior radiography; 3) lateral radiography; 4) tomography, and 5) oblique radiography, radiographies with contrast or other methods if required.

RESUMEN

El orden de la exploración radiológica de la hidatidosis pulmonar debe ser como sigue: 1) radioscopía simple; 2) radiografía postero-anterior o antero-posterior; 3) radiografía lateral; 4) tomografía, y 5) radiografía oblicua, radiografías con contraste u otros métodos cuando se requieran.

RESUME

L'ordre de l'exploration radiologique dans le cas de kystes hydatiques du poumon est le suivant: 1) radiographie standard; 2) radiographies postéro-antérieur ou antéro-postérieure; 3) radiographie latérale; 4) tomographies, y 5) radiographies obliques. Si c'est nécessaire, radiographies avec substance de contraste ou autres méthodes.

Tropical Eosinophilia Treated with ACTH

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The clinical condition which has been variously designated as tropical eosinophilia, eosinophilic lung, pulmonary eosinophilosis, and Weingarten's syndrome,[1] has been familiar to physicians engaged in chest work in South India even prior to 1930; however, it was only since the full description of the syndrome by Frimodt-Møller and Barton in 1940[2] that it has been recognized as a distinct entity and that many workers have been interested in investigating its etiology.

Etiology

Broadly speaking, there are two theories regarding the etiology of tropical eosinophilia: (1) an infection theory, possibly of virus origin, and (2) a theory favoring an allergic state, closely allied to bronchial asthma. Parasitic infections such as ascariasis, amoebiasis, filariasis, and acariasis have been incriminated from time to time with no adequate basis. We believe the disease is a manifestation of vascular allergy, the vessels of the pulmonary parenchyma being the basic shock tissue as compared to the bronchioles in simple and uncomplicated asthma. The remarkable increase in the total number of white blood cells and in the eosinophils, to 50 per cent or over, indicates that the hemopoietic tissues participate in the general allergic response. Apparently the bone marrow may be a shock organ which may react under allergenic stimulation by multiplication of its cellular elements as in the disease under discussion or, by suppression as in agranulocytosis. In other words, the asthmatic diathesis (Hurst) or the reaction pattern X (Rackemann) is an essential prerequisite. Of the possible variations in bronchial asthma, as depicted in Table I, type III corresponds to the usual case of tropical eosinophilia, and type IV, with its pleural and joint effusions, corresponds to the occasional case.

Symptoms and Signs

Low-grade pyrexia, cough with an asthmatic wheeze, anorexia, and loss of weight are the outstanding symptoms of this syndrome. Physical examination reveals the signs of an asthmatic bronchitis. A circulating eosinophil count of 2,000, as compared to a normal of 80 to 160 per cubic millimeter, is arbitrarily chosen as the minimum eosinophil count necessary for the diagnosis of this condition. In addition, roentgenograms

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TABLE I

TYPES OF BRONCHIAL ASTHMA . . THEIR CLINICAL SIGNS

Types of Bronchial Asthma	Blood Count		Mottling Roentgenographic	Other Findings
	W.B.C.	Per Cent Eosinophils		
I	Normal	5	Absent	——
II	Normal	5 to 30	Absent	——
III	Increased	Above 30	Present	——
IV	Increased	Above 30	Present	Pleural and/or Joint Effusions
V	Increased	Above 30	Present	Periarteritis Nodosa, Lesions in Brain, Abdomen, etc.

show various degrees of mottling; the densities are scattered diffusely through both lung fields, in contradistinction to the original Loeffler's syndrome[3] in which transitory, migratory bronchopneumonic patches occur with different areas being involved in succession rather than simultaneously. The roentgenologic appearance of a typical case of eosinophilic lung is observed in Figure 1. The distinction from miliary tuberculosis may be difficult; however, the individual opacities in tropical eosinophilia are larger ("pepper" compared to the finer "mustard"), and the uniform pleural haze present in the background of most roentgenograms of miliary tuberculosis is absent in tropical eosinophilia.

Rationale of Therapy

In view of the etiology of the disease, and of the known effect of ACTH in allergic states and in the envoked eosinopenic response, a series of patients with tropical eosinophilia were treated with ACTH. To our knowledge only one report has appeared in the literature, that of Rose et al,[4] wherein a single patient was given intramuscular administrations of ACTH with no satisfactory results. Our report presents a full description of 24 patients treated with ACTH administered by the intravenous route.

Selection of Patients

Of the 24 patients, ranging in age from 12 to 56, four were women. All presented the usual signs and symptoms of tropical eosinophilia; however, it is interesting to note that 10 had slight hemoptysis at some time or other in the course of their illness, and 12 had history of one or more previous similar attacks.

General Plan of ACTH Treatment

Each patient was observed for three days prior to commencement of treatment. ACTH was then given on 10 consecutive days, in doses of 5 I.U. on the first day, 10 I.U. on the second, 20 I.U. on the third through the eighth day, 10 I.U. on the ninth, and 5 I.U. on the 10th day. The daily dose of ACTH was added to 500 cc. of 5 per cent glucose, and administered intravenously over a period of eight hours. Excepting for the gradual increase and tapering off of this ACTH dosage, the schedule fol-

lows that of Renold et al.[5] A maximum intravenous ACTH dosage of 20 I.U. was also adopted by Mandel et al.[6] The patients were discharged two or three days following termination of therapy, and were requested to return a week later. All patients were then periodically observed for six to 12 months after treatment.

All were placed on a minimum sodium diet and in order to counteract hypokalemia, owing to the known increased potassium excretion with

ACTH IN TROPICAL EOSINOPHILIA

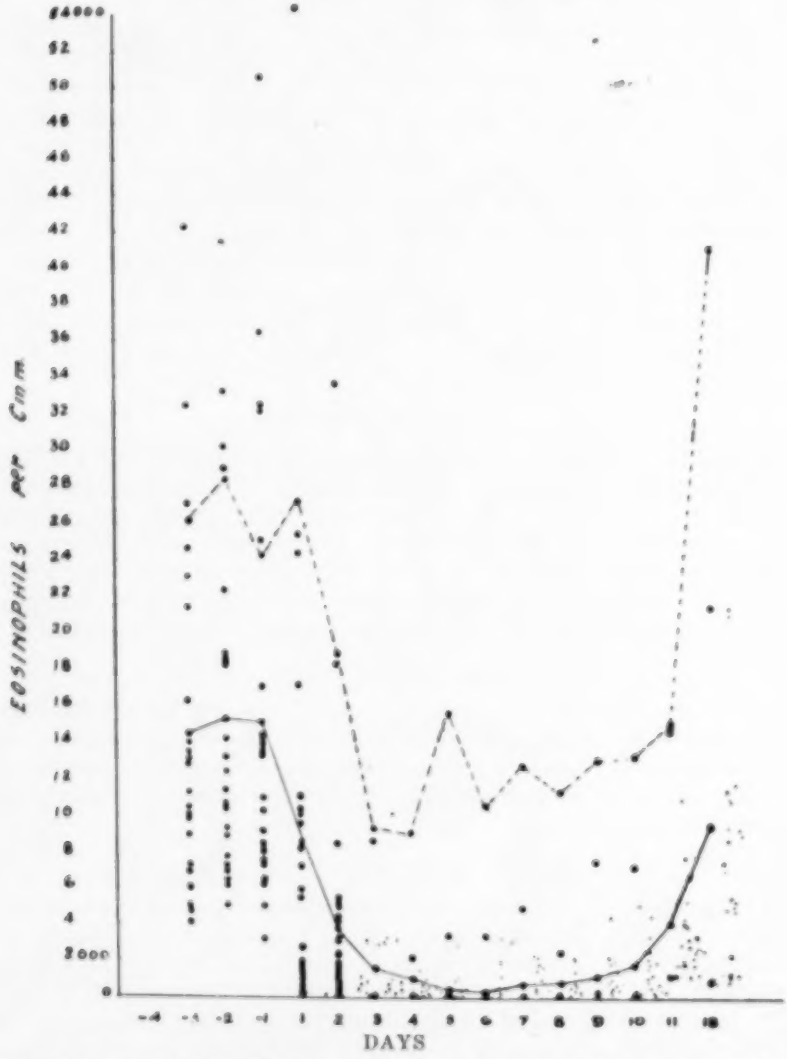

CHART 1: Average Eosinophil count changes in 24 patients, before, during, and after ACTH therapy (continuous line). Patient No. 17 showed poor response (dotted line).

TABLE II
SUMMARY OF ACTH EFFECTS
ON 25 PATIENTS WITH TROPICAL EOSINOPHILIA

		Pre-treatment					*Mid-treatment*					*Post-treatment*				
Case Number	*Sex and Age*	*Absolute Eosinophil Count*	*Urine 24 hour 17-ketosteroid mg.*	*E.S.R. Mms./hr.*	*Clinical State*	*X-ray Grading*	*Absolute Eosinophil Count*	*Urine 24 hour 17-ketosteroid mg.*	*E.S.R. Mms./hr.*	*Clinical State*	*X-ray Grading*	*Absolute Eosinophil Count*	*Urine 24 hour 17-ketosteroid mg.*	*E.S.R. Mms./hr.*	*Clinical State*	*X-ray Grading*
1	M-37	11,003	3.9	57	++	V	630	15	55	Nil	III	4,060	17.79	26	Nil	I
2	M-22	50,869	4.56	48	+	VI	3,554	5.61	33	Nil	IV	14,408	4.8	33	Nil	I
3	F-16	14,186	3.02	52	+	IV	412	2.73	31	Nil	II	3,150	5.75	47	Nil	I
4	M-17	8,407	8.45	72	++	VI	2,832	5.54	32	Nil	III	11,628	8.14	23	Nil	II
5	M-50	13,686	8.14	90	++	I	1,220	12.9	75	Nil	0	4,537	12.54	47	Nil	0
6	M-16	3,631	2.30	9	+	IV	0	3.93	8	Nil	II	2,662	2.49	4	+	II
7	M-27	6,362	3.57	19	++	III	80	7.8	10	Nil	I	1,911	6.84	5	Nil	I
8	M-14	6,272	2.93	45	+	III	169	7.78	21	Nil	0	925	4.59	19	Nil	I
9	F-40	17,647	4.675	10	+	IV	111	14.95	76	Nil	0	5,338	8.88	60	Nil	I
10	M-37	7,459	4.04	63	+	V	0	5.75	38	Nil	I	1,968	3.27	32	Nil	I
11	M-56	11,067	2.7	40	++	IV	50	10.87	24	Nil	II	1,232	5.43	25	Nil	0
12	M-14	10,575	3.41	29	++	IV	202	——	35	+	II	5,556	7.5	14	Nil	I
13	M-26	24,974	5.2	67	+	VI	193	11.63	67	Nil	II	7,872	4.47	56	Nil	I
14	M-13	13,944	10	45	+	V	202	11.63	70	Nil	I	3,145	1.15	26	Nil	I
15	M-22	9,391	8.46	49	++	I	1,170	10.34	59	+	0	3,403	1.66	40	+	0
16	M-36	8,596	4.39	87	+	V	200	7.74	63	Nil	II	1,300	6.27	51	Nil	II
17	M-30	24,141	3.41	65	++	IV	15,750	6.9	50	++	II	17,103	2.47	48	++	I
18	M-27	4,960	7.61	65	+	I	0	11.1	36	Nil	I	1,310	2.03	22	Nil	I
19	F-22	7,002	2.7	80	+	III	53	5.69	54	Nil	I	2,357	6.21	22	Nil	I
20	M-31	14,105	4.28	45	+++	III	1,630	8.37	32	Nil	I	2,599	2.2	—	Nil	II
21	M-22	36,826	5.5	50	++	III	583	7.65	35	Nil	0	7,533	5.28	31	Nil	0
22	M-36	32,146	2.76	83	+	II	1,488	2.79	69	Nil	0	2,886	4.03	47	Nil	I
23	M-20	32,363	3.06	28	+	III	145	4.28	10	Nil	I	7,139	13.58	9	Nil	I
24	M-23	8,294	3.76	8	++	III	378	3.52	8	+	I	3,578	3.81	5	Nil	I
25*	F-47	35,843	1.24	63	++	II	26,065	0.18	85	++	II	22,080	1.08	64	++	II

*This patient received 2 I.U. ACTH daily treatment schedule.

ACTH administration, they were given 60 to 90 grains daily of a potassium salt mixture.

The 2 I.U. Per Day Schedule: While this study was in progress, our attention was directed to the Cape, Carruthers, et al article[7] in which it was stated that the minimum effective intravenous "drip" dose of ACTH required to produce maximal eosinopenic response in the minimum time is 0.1 to 0.25 I.U. per hour administered over an eight hour period. Therefore, in Case 25 (Table II) ACTH was given in hourly doses of 0.25 I.U. totaling 2 I.U. during the eight hour slow intravenous drop period. This schedule was continued in Case 25 for 10 days with all observations maintained in the usual way. Compared with those receiving the usual dosage, no effective fall in the eosinophil count or rise in urinary 17-ketosteroids occurred in the post-treatment period. Furthermore, there was no remission in the clinical and radiologic appearances. It may be pointed out that the Cape et al patients who received this low ACTH schedule were those having rheumatoid arthritis and alopecia, conditions which are not usually associated with high blood eosinophil levels as noted in tropical eosinophilia. In addition, the absolute blood eosinophil values were not recorded by these workers.

Data Recorded: During the entire 15 or 16 day period, the following data were obtained: temperatures every four hours; daily weight; morning and evening blood pressure; daily fluid intake and output; daily urine, albumin, and sugar; plus white blood and eosinophil counts. All blood counts were done by the same observer and the blood was secured between 5 and 6 p.m. in order to avoid errors owing to diurnal variations in the eosinophil count. In each instance, the eosinophil count was done both

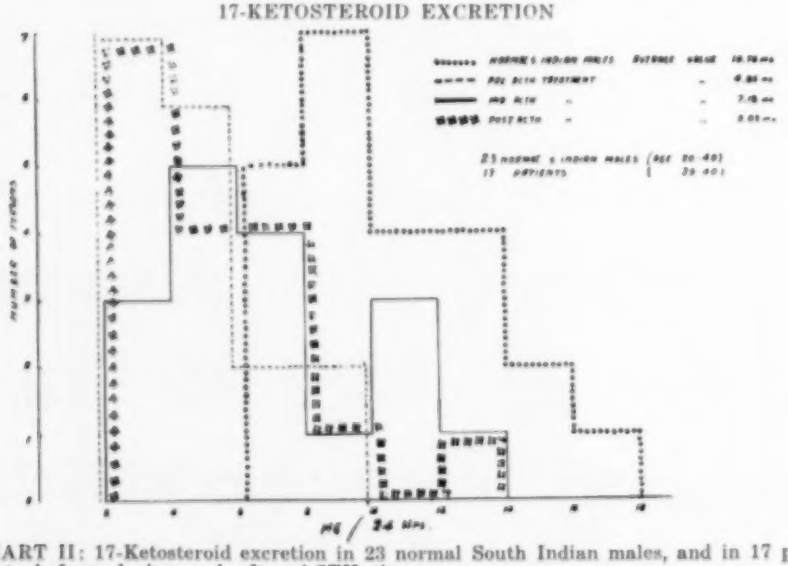

CHART II: 17-Ketosteroid excretion in 23 normal South Indian males, and in 17 patients before, during and after ACTH therapy.

by a direct method using Hinklemann's fluid (eosin yellow 0.5 Gm., phenol 0.5 cc. and formalin 0.5 cc. in 100 cc. of distilled water), counting two chambers, and by a differential count of blood smears counting 500 cells each time.

On three occasions, i.e. in the pretreatment, midtreatment, and posttreatment periods, the following data were obtained: photometric estimation of 24 hour excretion of urinary 17-ketosteroids using the Zimmermann reaction as advocated by the M.R.C. Committee on Clinical Endocrinology[8]; serum sodium and serum potassium levels; erythrocyte sedimentation rate using the Westergren's method; fasting blood sugar; roentgenogram of chest; and vital capacity. In addition, during the pretreatment observation period the motion was examined microscopically, the night blood was observed for microfilaria, the sputum for acid-fast bacilli, and the blood for Kahn and Wassermann reactions.

Results of Treatment

Of the 24 patients, 22 had satisfactory remission of symptoms and signs. Thirteen received relief on the second day, three on the fourth day, three on the sixth day, one on the seventh day, and two on the eighth day of the 10-day course of infusion. Thus the average patient was free of symptoms on the third day of treatment. Physical signs in the chest showed a corresponding or slightly delayed clearing. All expressed a general sense of well-being, and in two this bordered on a state of mild euphoria.

Apart from the two who had little remission of symptoms or signs during the course of therapy, three subjects experienced a return of symptoms and signs at the time of discharge, which was usually two days after discontinuation of therapy. In these instances, the symptoms required a longer period to disappear and the eosinopenic response was less marked than in the other patients. In one case "the rebound relapse," occurring within a week after discontinuation of therapy, was so severe that three additional infusions of ACTH had to be given.

Temperature: Two had temperatures of 101 F. during the observation period, and had a return to normal on the second day of therapy; their temperatures remained normal thereafter.

Weight: Twenty-one of the 24 had an increase in weight after completion of therapy. The average increase was 2.7 pounds, the most being five pounds and the least one pound. The other three maintained their weight.

Blood Pressure: This was recorded both in the morning and in the evening, i.e., before and after the infusion. In most instances the evening reading was slightly higher than that in the morning. Comparing the blood pressure values of the first observation day with the last posttreatment day, it was observed that there was no appreciable change in either the systolic or the diastolic readings, other than in the two cases described later.

Urine: Daily urinalysis revealed no albumin or sugar throughout the period of study.

Eosinophil Counts: The absolute eosinophil count per ccm. initially in six patients was 20,000 or over, in eight 10,000-20,000, and in ten 3,631-10,000. The highest eosinophil count was 50,869. (Table II)

Curiously in three the eosinophil count rose after the first infusion of 5 I.U., though subsequently with the continuation of therapy the count came down as in others. The number in whom the eosinophil count reached the minimum values between 0 and 400 per ccm. was 13. Chart I records the eosinophil counts in 24 before, during, and after treatment. It will be observed that following a marked drop in the count there was a tendency to rise with tapering off of ACTH dosage, and that within a week of cessation of treatment the counts definitely rose, usually to about half the initial values. Table III records the number having minimal eosinophil counts on each day of treatment.

The counts as obtained by the indirect differential method, and as recorded in Chart I, were always slightly higher than those obtained by the direct method. In the latter method there is a tendency on certain occasions of the eosinophils to clump and some of the cells are readily destroyed on shaking or standing, as mentioned by Swanson et al[9] in their discussion on the eosin-in-acetone method of staining eosinophils. A slight modification of this was used in our study. These destroyed cells would thus be lost in the direct count whereas they would be included in a differential count. However, only on 14 occasions of 336, was there a disparity of more than 10 per cent between the direct and the indirect methods. It should be stated that in our later studies the direct count was less time-consuming.

No changes were noted in the gross structure of the eosinophil.

Urinary 17-Ketosteroids: Chart II shows the trend in the values of the 17-ketosteroids excretion before, during, and after treatment, while columns 4, 9, and 14 of Table II give the actual figures in each of the cases. There was an increased 17-ketosteroid excretion during midtreatment in most instances, and the trend in values did not universally follow the change in the clinical course. Similar observations, both with excretion of 17-ketosteroids[10] and glucocorticoids[11] have been reported in asthma. The average value for 17-ketosteroid output per day before commencement of ACTH therapy was 4.70 mg., in the midtreatment it rose to 8.00 mg., and immediately post-treatment the value fell to 5.47 mg. These are average values of the whole group, disregarding age and sex; the few female and young patients did not excrete significantly lower amounts of 17-ketosteroids than the other patients.

It is of interest to note that the 4.70 mg. is much lower than the mean 24 hour 17-ketosteroid excretion in apparently normal persons of South Indian origin, as determined for purposes of comparison in the course of these investigations. The mean normal value in 23 men between the ages of 18 and 27 years was 10.81 mg.; this is significantly less than the mean accepted as normal in western subjects.[12] Mason and Engstrom,[13] in an extensive survey of western literature, give mean normal values between 12.5 and 16.7 mg. per day.

It is known that a chronic disease[14, 15, 16] reflects itself in excretion of lowered amounts of 17-ketosteroids, and that this has been reported in asthma,[10, 11] a condition related to tropical eosinophilia.

Serum Sodium: There was no appreciable change in the readings before, during, and after treatment.

Serum Potassium: A small but definite fall in the serum potassium occurred in all patients despite the administration of oral supplements of potassium to preclude the possibility of hypokalaemia.

TABLE III
MINIMAL EOSINOPHIL COUNT ON EACH DAY OF THERAPY

Day of Therapy	*Number of Cases*
1st	0
2nd	0
3rd	2
4th	5
5th	8
6th	5
7th	3
8th	0
9th	1

Erythrocyte Sedimentation Rate: Table II, columns 5, 10, and 15, gives the erythrocyte sedimentation rate values. Blood was always secured at eight in the morning, before breakfast and onset of tropical heat. The average initial value was 51.5 mm. in one hour, the midtreatment value 41.3 mm., and the immediate post-treatment value 29.7 mm. Thus there was a gradual fall in the sedimentation rate as a result of ACTH therapy, and this fall corresponds to the reduction in eosinophils and in symptomatology. Fearnley and Bunim[17] mention that the fall in sedimentation rate results from a nonspecific factor of ACTH; four of their seven normal subjects had a fall in sedimentation rate and in plasma fibrinogen following ACTH administration. In view of this latter observation, it is doubtful that the sedimentation rate is of value as an index of improvement of the morbid process in tropical eosinophilia during ACTH therapy.

Fasting Blood Sugar: The average pretreatment fasting sugar level in the 24 patients was 74.2 mg. per 100 cc. of blood. Midtreatment readings averaged 92.1 mg., and post-treatment 69 mg.

Roentgenograms: Roentgenograms of each case taken before, during, and after treatment were classified into seven grades, ranging from 0 to VI, according to extent of distribution of mottling, and density of opacity. Table II, columns 7, 12, and 17, shows that in every case there was appreciable improvement in the roentgenographic appearances as a result of treatment. Figures 1, 2, 3, and 4 cover roentgenograms taken before and after treatment in two.

Vital Capacity: The average value of the vital capacity measurement prior to treatment was 1199 cc., and after treatment 1443 cc. Our impression, however, is that the values are likely to be higher if a correction is made for the "instrumental factor" in the measurements. According to Daniel S. Lukas[18] ACTH and cortisone have sustained bronchodilating action, and decrease the reactivity of the bronchial musculature to allergens. It is likely that the improvement noticed in the vital capacity in these patients with tropical eosinophilia treated with ACTH is due to a diminution in the eosinophilic exudation into the alveolar spaces as well as to the release of bronchial musculature spasm. It may be noted that these vital capacity values are far below mean normal values in western subjects and below the mean normal values in South Indian subjects.[19]

Other Laboratory Data: Three patients showed Ascariasis, while none showed endamoeba histolytica cysts, or microfilaria in the night blood. One of the 24 had a positive Wassermann reaction.

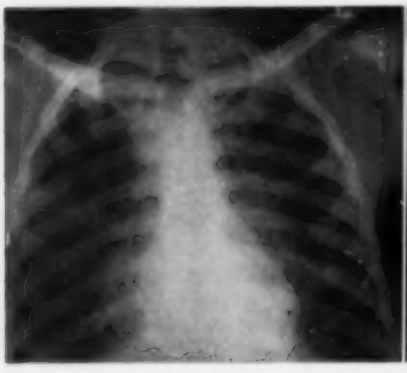

FIGURE 1

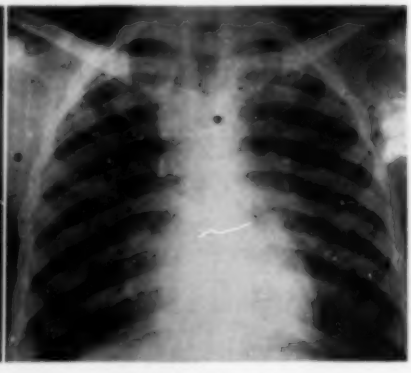

FIGURE 2

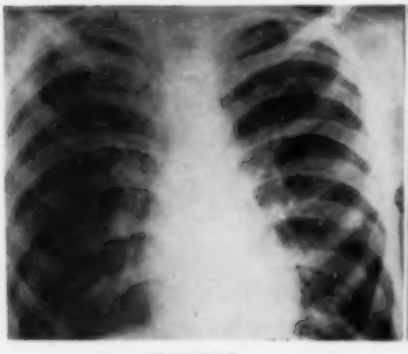

FIGURE 3

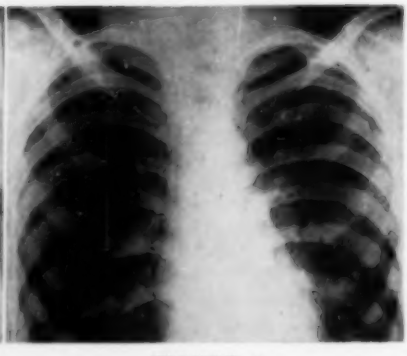

FIGURE 4

Figure 1: Typical skiagram with extensive mottling in tropical eosinophilia.—*Figure 2:* Same case as in Figure 1, however, following ACTH therapy.—*Figure 3:* Case No. 14 before ACTH therapy. *Figure 4:* Same case as in Figure 3; however, following ACTH therapy.

Side Effects

Three complained of headache at the end of each day's infusion, for the first few days, and one had persistent giddiness on the last three days. The headache and giddiness were not associated with rise in blood pressure. Two had substernal pain during the latter half of treatment, and one had mild pruritus and bullae around site of infusion on the seventh day. Fullness of the cheeks to an obvious degree were observed in two. None of the four women developed acne, hirsutism, or disturbance of menstruation.

Two Cases Wherein ACTH had to be Discontinued

ACTH was discontinued in two, one of whom had received the 2 I.U. per day schedule, approximately a month previously. Initially there was no albuminuria or hypertension; however, with the appearance of a trace of albumin on the second day of therapy, ACTH was discontinued and the patient was observed for the following two days. Since albumin disappeared during this period, the patient received for the next two days 10 I.U. and 20 I.U. of ACTH, according to the usual schedule, with reappearance of albumin. Although the blood pressure was not raised, ACTH was discontinued.

In the second case, ACTH was discontinued because of albuminuria and elevation of both systolic and diastolic blood pressure (Table IV).

TABLE IV
EFFECT OF ACTH ON BLOOD PRESSURE AND URINE IN 1 PATIENT

Day	*ACTH Daily Dose*	*Blood Pressure*		*Urine Albumin*
		Morning	*Evening*	
—2		——	——	——
—1		——	120/80	Negative
1	5 I.U.	134/94	160/106	Negative
2	10 I.U.	160/112	172/112	Negative
3	20 I.U.	170/106	164/102	Negative
4		176/118	180/116	Trace
5		140/96	164/118	Trace

Discussion

Our studies on tropical eosinophilia indicate that ACTH is definitely effective and that suppression of clinical and hematologic manifestations of the disease are temporary. Relapses occur so early after treatment and so invariably that the regular use of ACTH as a therapeutic agent is not advocated. It has, however, a definite place in the treatment of the severe case with status asthmaticus. Our findings are in contrast to those of Rose et al[4] who did not observe an appreciable eosinopenic response with intramuscular administration of ACTH in the one patient of tropical eosinophilia studied.

We have also to remember the one with a trace of albuminuria and hypertension, and the one with a trace of albumin alone in whom therapy

were discontinued. ACTH has been reported to have induced hypertension in subjects with a variety of diseases; however, this has been the exception rather than the rule. Plotz[20] reported hypertension and subarachnoid hemorrhage developing after ACTH in the treatment of disseminated lupus, and Gordon and McLean[21] observed transient hypertension during the use of ACTH for the treatment of ocular conditions.

The spectacular fall in the eosinophils and the rise in 17-ketosteroids as a result of ACTH administration should support the concept that tropical eosinophilia is a collagen disease or a state of allergy. On the basis of a more than 12-month follow-up study of a number of patients, we believe that an elevated eosinophil count invariably persisted in those subjects who never showed the normal percentage of eosinophils. From time to time, there is an exacerbation with considerable increase of eosinophils preceding the well-known symptoms of the disease. We believe, therefore, that tropical eosinophilia represents an allergic state, dependent partly on the host himself and partly on unknown factors in the environment. It is also suggested that bacterial allergy on the basis of chronic upper respiratory infection or mild bronchiectasis, together with extrinsic allergens, is responsible for the unusual eosinophilic response.

Biochemical studies, to establish the inter-relationship between the following, will aid in providing the answer to the etiology of tropical eosinophilia:—histidine, histamine, the eosinophil cell, Charcot Leyden crystal, spermin phosphate, ACTH, and hyaluronidase. Although there is considerable literature on these subjects, no exact knowledge is available on their mutual relationships.

We do not believe that arsenic "is a cure" for tropical eosinophilia as has been held by some writers. It produces a remission variable in duration, and we have seen several tropical eosinophilia patients with arsenical reactions, including fatal encephalopathy following a second or more course of therapy. These indicate that the subjects were sensitized to arsenic. During the last four years, hundreds of cases have been treated on the usual antiallergic program and without arsenic, with satisfactory results.

SUMMARY

Data regarding eosinophil counts, 17-ketosteroids excretion, and other clinical and biochemical observations made during the treatment of 24 patients of tropical eosinophilia with ACTH by the intravenous route are presented.

It is concluded that ACTH has a definite though limited place in the treatment of this condition. The pathogenesis of tropical eosinophilia is briefly reviewed and suggestions for future studies made.

RESUMEN

Se presentan datos respecto de la cuenta de eosiinófilos, excreción de 17-ketosteroides y otras observaciones clínicas y bioquímicas son presentados después del tratamiento de 24 enfermosde eosinofilia tropical con ACTH.

Se concluye que la ACTH tiene un lugar definido aunque limitado en el tratamiento de esta afección. La patogenia de la eosinofilia tropical se revisa de modo breve y se hacen sugestionessobre futuros estudios.

RESUME

Les auteurs rapportent une série de documents concernant le taux de l'éosinophilie sanguine, l'excrétion des 17-cétostéroïdes, ainsi que d'autres constatations cliniques et biochimiques qu'ils ont pu effectuer au cours du traitement par l'A.C.T.H. intra-veineuse de 24 malades atteints d'éosinophilie tropicale.

Les auteurs en tirent la conclusion que, bien que limitée, l'action de l'A.C.T.H. est incontestable dans le traitement de cette affection. Ils passent en revue rapidement la pathogénie de l'éosinophilie tropicale et proposent quelques projets d'études sur ce sujet.

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Tropical Eosinophilia Treated with Cortisone

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Twenty-four subjects of tropical eosinophilia were treated with ACTH and a detailed paper[1] giving the methods employed and the results already have been presented in this Journal. The present study to evaluate the place of cortisone in the treatment of tropical eosinophilia was planned on entirely similar lines.

Cortisone acetate (Cortone, Merck) was administered orally over a 10-day period after a preliminary two-day period of observation in the following schedule:

First day	50 mgms.
Second day	75 mgms.
Third to eighth day both inclusive	100 mgms.
Ninth day	75 mgms.
Tenth day	50 mgms.

The cortone was administered in the form of 25 mgm. tablets equally distributed during the 24-hour period. The usual precautions in the selection of cases and in the treatment to counteract possible hypokalaemia were taken. The various observations mentioned in the previous paper were maintained. The direct method of counting the eosinophils was employed throughout the study.

Table I gives a summary of the more important data concerning the 21 subjects of tropical eosinophilia who formed the subjects of this study.

Clinical Response

Of the 21 subjects, 19 showed satisfactory remission of symptoms and signs as a result of the treatment. One of the two cases showing little or no response had shown a similar lack of effect to ACTH in the previous study. Thirteen cases had become symptom-free by the third day of treatment, while the other eight took up to five days more. The duration of clinical remission induced by cortisone was not longstanding and was similar to the remissions induced by ACTH.

Side Effects

Seven subjects complained of pain in the chest of a vague nature, not confined to any local area. This subjective symptom of pain, not present previously, was rather surprising in view of the analgesic effect of cortisone. No case showed albuminuria or elevation of blood pressure, complications noted in the previous study with ACTH.

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Our thanks are due to Merck & Co. for the free supply of cortone used in these studies. The research was financed by the Madras State Research Fund to whom we are indebted. Mr. S. Guha, Assistant Professor of Statistics, Presidency College, helped us with statistical advice.

TABLE I

Case Number	Age and Sex	Pre-treatment		Mid-treatment		Post-treatment	
		Absolute Eosinophil Count.	X-ray Grading	Absolute Eosinophil Count.	X-ray Grading	Absolute Eosinophil Count.	X-ray Grading
I	M-37	21,450	IV	3,232	II	5,400	I
II	M-22	5,476	II	1,650	I	4,150	I
III	M-22	13,950	III	9,400	III	7,250	II
IV	M-23	11,950	V	2,150	II	4,600	II
V	M-22	10,200	V	2,400	I	7,200	II
VI	M-20	14,850	II	575	II	2,000	I
VII	M-22	16,850	III	9,250	I	11,100	II
VIII	M-28	9,000	III	5,025	I	2,760	I
IX	M-30	30,150	V	330	II	1,700	I
X	M-29	9,200	IV	2,150	II	5,125	II
XI	M-17	8,250	II	200	II	1,575	II
XII	M-21	18,625	VI	3,775	II	3,075	II
XIII	M-46	12,525	III	2,375	I	4,000	II
XIV	M-27	9,650	III	1,575	II	4,075	II
XV	M-30	31,600	II	3,300	I	2,100	I
XVI	M-19	15,600	IV	1,275	IV	3,375	IV
XVII	M-25	21,075	IV	1,525	II	1,675	III
XVIII	M-28	24,925	IV	375	IV	450	IV
XIX	M-29	7,950	IV	5,700	II	1,250	III
XX	M-21	4,775	IV	4,250	I	750	II
XXI	M-23	20,025	III	5,625	III	4,025	II

Urinary 17-Ketosteroids

The average values in milligrams per 24 hours of the neutral 17-ketosteroids in the urine were—

Pre-treatment	7.51
Mid-treatment	7.45
Post-treatment	8.54

It will be noticed that the distinct rise in the mid-treatment stage ocurring during ACTH medication is absent during cortisone treatment. This behaviour of the 17-ketosteroid excretion is as expected with cortisone therapy. The question of 17-ketosteroid excretion in normal South Indian subjects compared to those suffering from tropical eosinophilia has been dealt with in a separate paper[2] from our laboratories.

Erythrocyte Sedimentation Rate

Table II shows the mean E.S.R. values in millimeters at the end of one hour by Westergren's method both in the cortisone series and in the ACTH series. It will be noted that the fall in the E.S.R. is more appreciable in the cortisone series.

TABLE II

	Pre-treatment	Mid-treatment	Post-treatment
Cortisone Series	42.8	25	18
ACTH Series	51.5	41.3	29.7

Eosinophil Counts

A comparison of the mid-treatment values in the absolute eosinophil count shows that in 15 of the 24 ACTH cases, a figure about 400 or below was reached, while such low figures were noticed only in three of the 21 in the cortisone series. This given the apparent impression that ACTH may be more effective in bringing the absolute count to near normal levels. However, the mean fall between the pre-treatment and mid-treatment values was calculated in the two series. The mean fall with ACTH (24 cases) was 14452 while with cortisone (21 cases) the fall was 11997. Applying the 't' test to these figures, the result 0.81 (43 degrees of freedom) is not significant.

It has already been mentioned that in both the series there has been a rise from the mid-treatment to the post-treatment values. The mean rise in the ACTH series was 3606 (all cases showing positive values) while in the cortisone series it was only 547.5 (7 showing negative values and 14 positive values). The value of 't' (43 degrees of freedom) is 3.9 which is significant, indicating that the "eosinophil escape" is more with ACTH than with cortisone.

Roentgenograms

Columns 4, 6 and B of Table I show the radiological improvement noted with cortisone treatment. For purposes of comparison cases showing x-ray grades IV, V and VI at the commencement of treatment were added together and it was examined how many of these showed a grade of I or O at the end of treatment. In the ACTH series, of 13 cases revealing such extensive mottling, 10 had shown excellent clearing of lesions, while in the cortisone series, of the 11 in these higher grades, only 2 presented excellent clearing.

DISCUSSION

The results of the use of cortisone in the treatment of tropical eosinophilia were very similar to those observed with ACTH. Neither drug could be offered as a permanent cure for the condition, but both produced immediate and appreciable improvement in the condition of the patient. The variations in the end results noted above appear to warrant the suggestion that the best results may be expected by starting treatment with ACTH and after five days, maintaining the treatment with oral cortisone for a longer period.

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Case Report Section

Right Aortic Arch Versus Mediastinal Tumors and Densities: Diagnostic Problems

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Difficulty in the differential diagnosis of tumors of the mediastinum is well known. Occasionally a right aortic arch may be the cause of a mediastinal density. In other cases mediastinal densities and tumors may simulate the picture of right aortic arch. Prompt diagnosis is essential if definitive treatment is to be instituted for diseases of the lymph nodes. This article is presented to emphasize the occasional close resemblance of right aortic arch and lymph node pathology and to stress the relative ease with which the two conditions can be differentiated in the office.

A right aortic arch may be variable in its course and configuration. When associated with a left descending aorta, the former will displace the esophagus and trachea to the left and anteriorly. This is borne out by posterior-anterior, oblique and lateral x-ray films respectively. However, when a right aortic arch is combined with a right descending aorta, the esophagus and trachea are displaced to the left and not anteriorly. If additional vascular anomalies coexist, angiocardiographic studies may be necessary.

In early childhood, tracheal displacement by a right aortic arch may occasionally give rise to upper and lower respiratory tract infections. In adult life, such displacement occasionally causes dysphagia, hoarseness or cough. The cases illustrated below were asymptomatic.

Case 1: J. P., a 53 year old male, showed on a routine survey chest x-ray film, a slightly enlarged heart and a somewhat dilated and elongated aorta. In the right hilar region there was a large dense semicircular shadow bordering medially on the cardiac and aortic silhouette. This density could have represented part of a tortuous aorta, aortic aneurysm, mediastinal tumor or enlarged lymph node. He was fluoroscoped with the aid of a barium drink to investigate the dense hilar shadow. During this examination a right-sided aortic arch with a left descending aorta was discovered accidentally. In the posterior-anterior chest film the aorta was displaced to the left and in the right anterior oblique position the right aortic arch displaced the esophagus and the trachea considerably forward (Figs. 1A and 1B). The right hilar density was part of the tortuous aorta. This case demonstrates how difficult the diagnosis of right aortic arch may be when the routine chest film shows the aortic shadow to be widened, elongated and tortuous.

Case 2: F. H., a 52 year old female, was referred to the clinic because of a slightly enlarged heart. She complained of fatigue. The chest x-ray film in the conventional posterior-anterior position showed a somewhat elongated and widened mediastinal aortic silhouette. The right upper portion of this shadow seemed to be the border of a small azygos lobe. Fluoroscopic examination with the aid of a barium drink was done to investigate the upper mediastinal structure. A right aortic arch with a left descending aorta was demonstrated. In the posterior-anterior x-ray film, the esophagus was displaced to the left and considerably displaced anteriorly in the right anterior oblique view (Figs. 2A and 2B). In this case the right aortic arch again was lost in the general dilatation and increased density of the upper mediastinal aortic shadow.

From the Bedford Chest Clinic of the Bureau of Tuberculosis, New York City, Department of Health.

Case 3: M. S., a 52 year old female, gave a history of antibacterial therapy and a short period of pneumoperitoneum for tuberculosis of the left upper lobe. The chest x-ray film demonstrated fibrosis in the apical-posterior segment of this lobe. The aorta was dilated and elongated. In the search for a possible cavity in the left apical region tomograms were done, in one of which a large rounded shadow was seen in front and somewhat to the right of the vertebra. (The film is not show here.) This gave the appearance of an enlarged mediastinal tuberculous lymph node. However, a possible right aortic arch was also considered as a diagnostic possibility. An esophagram confirmed the latter opinion. The posterior-anterior chest film demonstrated the esophagus to be displaced by the right aortic arch to the left (Figs. 3A and 3B). In the right anterior oblique position the aortic arch displaced the esophagus and trachea anteriorly, this being diagnostic of right aortic arch with left descending aorta. This case demonstrates the diagnostic and differential diagnostic importance

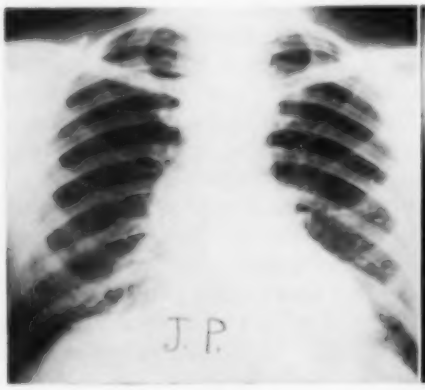

FIGURE 1A

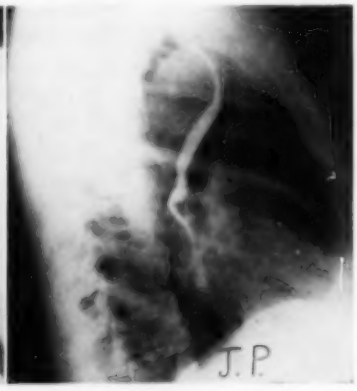

FIGURE 1B

Figure 1 (CASE 1)—J. P.: Right aortic arch with left descending aorta. Aortic tortuosity causing right mediastinal density. (*A*) P. A.—Large dense semicircular shadow in right mediastinum. (*B*) Right lateral view—Esophagram: Aorta displaces esophagus and trachea markedly forward.

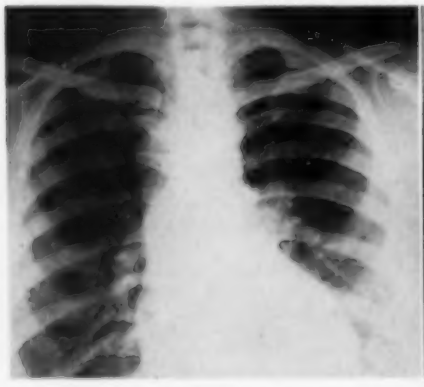

FIGURE 2A

FIGURE 2B

Figure 2 (CASE 2)—F. H.: Right aortic arch with left descending aorta-azygos lobe. (*A*) P. A.—Heart slightly enlarged to left. Mediastinal aortic silhouette somewhat widened and elongated, its right upper border fusing with azygos lobe. (*B*) Right Lateral View—Retro-esophageal aorta displaces esophagus and trachea considerably forward.

of this vascular anomaly. After the diagnosis of an enlarged tuberculous lymph node was excluded no further therapy was indicated.

Case 4: E. W., a 53 year old female, showed on routine survey examination a double lobed bilateral upper mediastinal shadow. Its rounded and sharply outlined right upper portion slightly overlapped the proximal part of the right clavicle, extended downward in a medial direction and merged with the cardiac silhouette in the region of its base. Its rounded and sharply outlined left upper part extended from the proximal end of the left clavicle and merged with the aortic shadow (Fig. 4A). The esophagram showed the esophagus to be displaced to the left and anteriorly and was

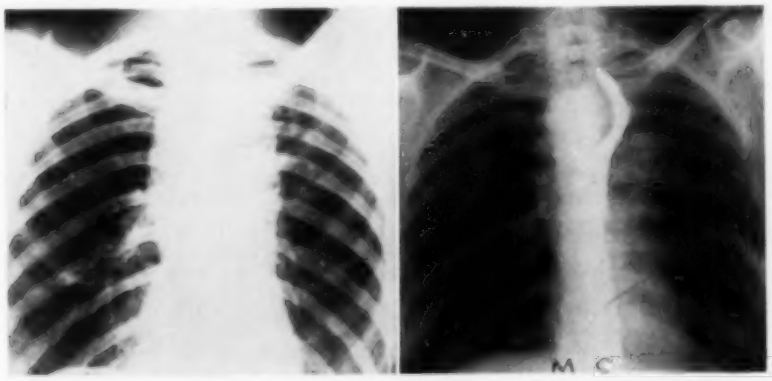

FIGURE 3A FIGURE 3B

Figure 3 (CASE 3)—M. S.: Old Healed Pulmonary Tuberculosis—Right aortic arch with left descending aorta. (*A*) P. A.—Pulmonary Fibrosis in apical-posterior segment of left upper lobe; aorta generally dilated and elongated. (*B*) Esophagram P. A. x-ray—The esophagus is considerably displaced to the left by a round, dense mediastinal shadow, representing a right aortic arch.

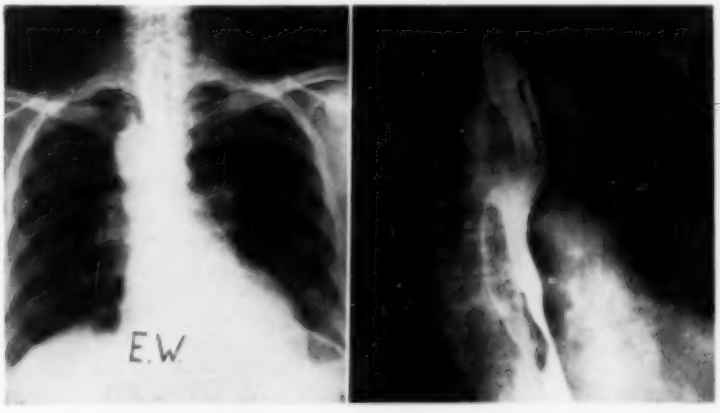

FIGURE 4A FIGURE 4B

Figure 4 (CASE 4)—E. W.: Right aortic arch with left descending aorta. (*A*) Esophagram P. A. x-ray—Double lobed, bilateral upper mediastinal shadow. The esophagus is markedly impinged on its right border and displaced to the left by the right aortic arch, represented by the right mediastinal density. The density in the left mediastinum has an osseous appearance and a dense bony border and represents the left side of the manubrium sterni. (*B*) Esophagram right anterior oblique position—The esophagus is displaced anteriorly by the right aortic arch.

diagnostic of a right aortic arch with a left descending aorta (Fig. 4B). The left part of the upper mediastinal density revealed an osseous structure and represented the left upper portion of the manubrium sterni.

Case 5: H. R., a seven year old girl was admitted to the Bedford Chest Clinic in February 1953. The routine chest film showed fibrotic changes in the lateral segment of the right middle lobe, and a double lobed bilateral upper mediastinal density (Fig. 5) similar to that seen in Case 4. The Mantoux test was 2 plus. The search for acid fast bacilli was negative. The esophagram was normal. The right upper part of the mediastinal shadow had an osseous appearance and structure on close study. It was therefore considered to be the right upper portion of the sternal manubrium. The left part of the mediastinal density proved to be the normal aortic arch. Enlargement of the lymph nodes was thus excluded and since subsequent chest films showed no changes, no further treatment was indicated.

Case 6: H. K., a 32 year old male, was admitted complaining of moderate productive cough. The routine chest x-ray film of January 8, 1953, revealed two right mediastinal densities, one in the superior mediastinum and the other below the right clavicle (Fig. 6A). The history disclosed that the patient had bilateral cervical lymph adenopathy and a biopsy had revealed typical tuberculous caseation necrosis.

He was placed on isonicotinic acid hydrozide and para-aminosalicylic acid. On October 15, 1953, the chest film showed resolution of the superior mediastinal density (Fig. 6B). The remaining lower density could have been suspected to be a right

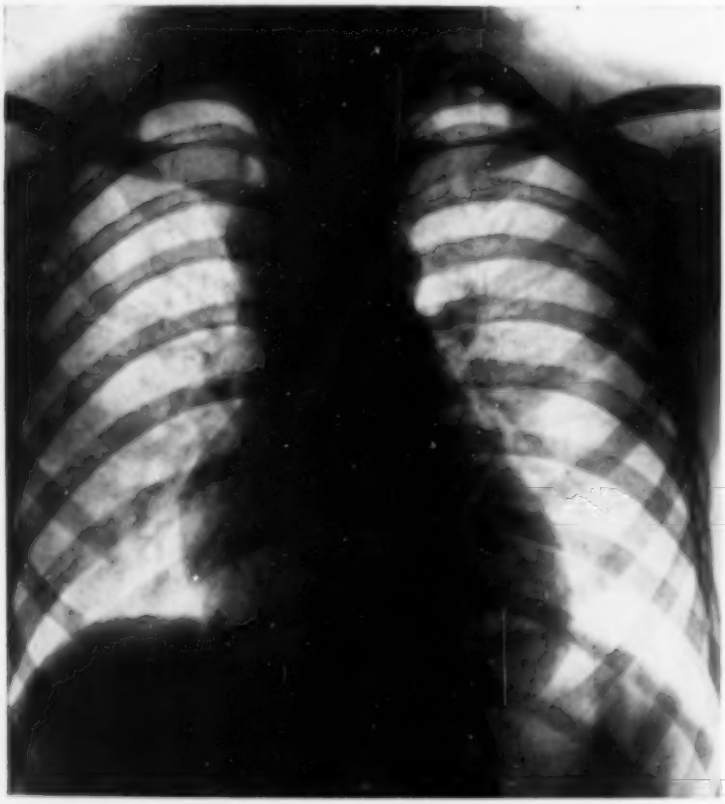

FIGURE 5 (CASE 5)—H. R.: Healed tuberculosis, lateral segment of right middle lobe. Double lobed, bilateral upper mediastinal density. The esophagram was normal. The right upper part of the density has an osseous structure and represents the right side of the manubrium sterni. The left and lower part of the mediastinal density proved to be the normal aortic knob.

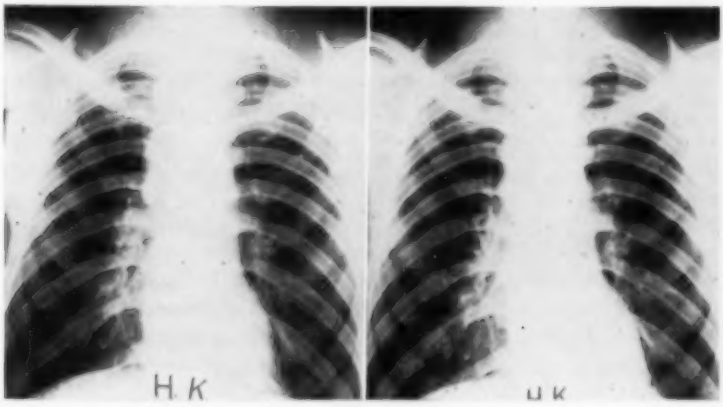

FIGURE 6A FIGURE 6B

Figure 6 (CASE 6)—H. K.: Enlarged Tuberculous Lymph Nodes in Right Upper Mediastinum. (*A*)—Two right mediastinal densities diagnosed as enlarged tuberculous lymph nodes. (*B*)—Resolution of the upper mediastinal density following antituberculous therapy. Lower density persists simulating right aortic arch.

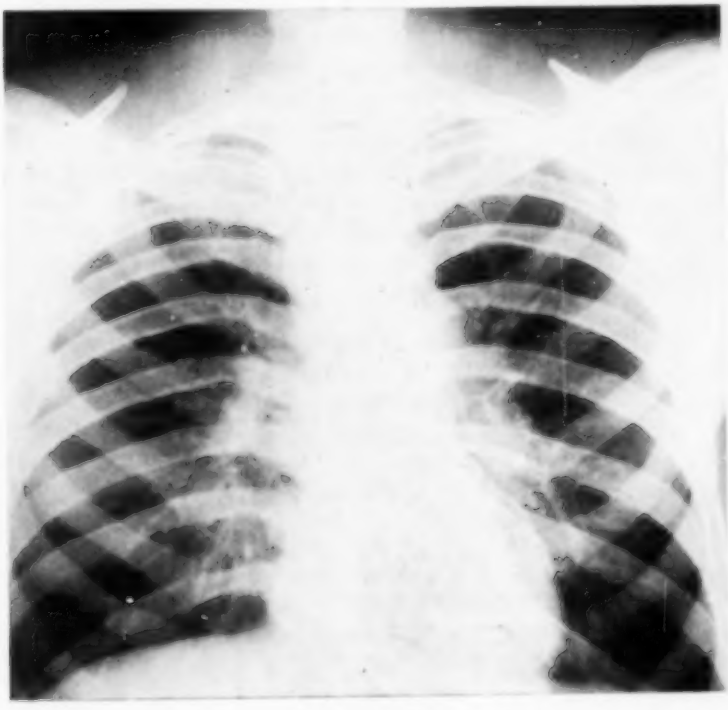

FIGURE 7 (CASE 7)—J. S.: Boeck's Sarcoid. Enlarged lymph node in right upper mediastinum simulating right aortic arch.

aortic arch. Fluoroscopic examination with a barium drink revealed the esophagus and trachea in normal position. After the diagnosis of right aortic arch had been excluded, continuation of the therapy was indicated.

Case 7: J. S., a 28 year old male, was admitted complaining of cough. The routine chest x-ray film showed a mediastinal density suggestive of right aortic arch (Fig. 7). On physical examination there was, however, generalized enlargement of the lymph nodes. A small cervical lymph node was removed for biopsy. Histological examination revealed Boeck's Sarcoid. The mediastinal lymph node which simulated the picture of right aortic arch was seen to disappear on subsequent films.

SUMMARY

It is of importance to recognize a right aortic arch not only for the occasional symptoms which may be produced by it, but more so, to differentiate it from mediastinal tumors and diseases of lymph nodes. The seven selected cases presented in this article, emphasize the diagnostic and differential diagnostic problems that we have encountered.

Three cases were presented in which the investigation for mediastinal and pulmonary pathology revealed a right aortic arch as an accidental finding. These three patients had somewhat dilated, elongated and tortuous aortic vessels. Their routine posterior-anterior chest films were not suggestive of right aortic arch. Two cases presented a bilateral upper mediastinal density suggestive of right aortic arch with additional mediastinal pathology. The esophagram revealed a right aortic arch in one case and excluded that diagnosis in the other. In both cases the manubrium sterni added to the diagnostic problems encountered. Positional deviations of the sternal manubrium are not infrequent. Two cases were shown with enlarged mediastinal lymph nodes simulating right aortic arch.

SUMARIO

Es de importancia el reconocimiento de un arco aórtico derecho, no sólo por los síntomas ocasionales que puede producir, sino todavía más para diferenciarlo de tumores mediastinales y enfermedades de los ganglios linfáticos. Los siete casos selectos presentados en este artículo, enfatizan los problemas de diagnóstico y de diagnóstico diferencial que nosotros hemos encontrado.

Se presentaron tres casos en los cuales la investigación por patología mediastinal y pulmonar reveló un arco aórtico derecho comohallazgo accidental. Estos tres pacientes tuvieron vasos aórticos algo dilatados, elongados y tortuosos. Sus placas de tórax de rutina postero-anteriores, no sugerían un arco aórtico derecho. Dos casos presentaron una sombra mediastinal superior bilateral sugerente dearco aórtico derecho con patología mediastinal adicional. El esófago grama reveló un arco aórtico derecho en uno de los casos y excluyotal diagnóstico en otro. En ambos casos el problema del manubrio esternal se agregó a los problemas diagnósticos encontrados. No son infrecuentes las desviaciones de posición del manubrio esternal. Doscasos tuvieron ganglios linfáticos mediastinales que simulaban arco aórtico derecho.

RESUME

Il est important de savoir reconnaitre une persistance de l'arc aortique droit, non seulement à cause des symptômes éventuels qu'il peut provoquer,

mais aussi pour le différencier des tumeurs et des affections ganglionnaires médiastinales. Dans les sept cas choisis es présentés dans cet article, les auteurs insistent sur l'importance des problèmes de diagnostic et de diagnostic différentiel auxquels ils se sont heurtés.

Ils présentent trois cas dans lesquels les examens pour diagnostiquer une affection médiastinale et pulmonaire révélèrent par hasard une persistance de l'arc aortique droit. Les trois malades avaient des vaisseaux aortiques quelque peu dilatés, allongés et sinueux. Les radiographies antéro-postérieures de contrôle ne faisaient pas penser à un arc aortique. Deux cas présentaient une ombre médiastinale bilatérale supérieure, évoquant un arc aortique avec une affection médiastinale surajoutée. L'opacification de l'oesophage révéla un arc aortique droit dans un cas, et dans un autre en exclut la possibilité. Dans ces deux cas, l'ombre du manubrium sternal compliqua les problèmes diagnostiques rencontrés. Des déviations de la position du manubrium sternal ne sont pas rares. Les auteurs en montrèrent deux cas, avec ganglions médiastinaux augmentés de volume, évoquant un arc aortique droit.

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Capillary Hemangioma of the Lung*

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Cavernous hemangioma of the lung synonymous with arteriovenous fistula or aneurysm of the lung is generally considered to be a congenital abnormality. According to Lindskog and Liebow,[1] at least 60 cases are in the literature. Roentgenologically, cavernous hemangioma has the appearance of a lobular shadow of increased density continuous with hilar vascular shadows. Clinically it is usually associated with polycythemia, cyanosis, clubbing of the fingers and dyspnea. A diagnosis is ordinarily made preoperatively and the indications for surgical excision are hemoptysis or dyspnea and, in the asymptomatic patient, the prevention of serious vascular complications.

As distinguished from cavernous hemangioma, capillary hemangioma of the lung presenting itself as a "coin" lesion and unaccompanied by any of the findings characteristic of cavernous hemangioma is an extremely rare lesion.[2, 3] The Chest Tumor Registry of the Armed Forces Institute of Pathology[4] has in its files only a few true capillary hemangiomas of the lung. Textbooks on pathology and thoracic surgery make no

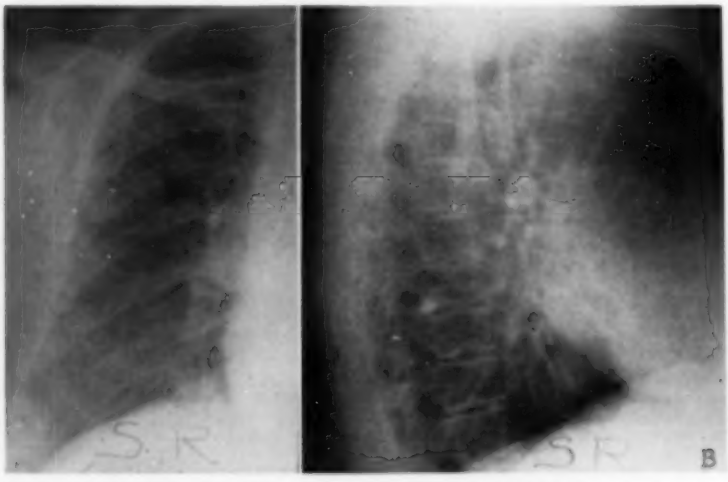

FIGURE 1A FIGURE 1B

Figures 1A and 1B: Preoperative roentgenograms showing the "coin" lesion within the lower lobe of the right lung adjacent to and immediately posterior to the heart shadow.

*Presented in part in the X-ray Symposium, 19th Annual Meeting, American College of Chest Physicians, New York, New York.

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mention of it and a review of reports dealing with pulmonary "coin" lesions revealed only a single case of unstated size in a 65-year-old female whose symptoms consisted of cough and expectoration and who was treated by lobectomy.[5] One instance without a statement as to the size of the lesion or the coexistence of an arteriovenous fistula was reported in 1944;[6] however, in view of the fact that it was treated by pneumonectomy it is likely that it was a cavernous rather than a capillary hemangioma presenting itstelf as a coin lesion. Another lesion described as an asymptomatic solitary round lesion was excised and found to be an hemangioma;[7] however, no statement was offered describing the lesion as cavernous or capillary.

The purpose of this communication is to report an instance of capillary hemangioma of the lung occurring as a "coin" lesion and treated by excision in order to obtain a diagnosis as well as relief from symptoms.

Case Report

S. R., a 59 year old white female, was first seen in March 1950 because of cough and expectoration of small quantities of mucoid and occasionally bloody sputum for several months. Significant physical findings included overweight, enlargement of both hands without edema and hypertension of 240/120. Urinalysis, blood count, blood urea nitrogen and electrocardiogram were within normal limits. Sedimentation rate was rapid.

Chest roentgenograms between 1950 and 1952 revealed no change in the calcium-free "coin" lesion within the lower lobe of the right lung at the cardiophrenic angle (Figures 1A, 1B and 2A). Bronchoscopy in 1950 and 1952 was noncontributory and examination of bronchial secretions revealed no tumor cells.

On August 1, 1952 right lower lobectomy was performed.* Recovery was uneventful and she became free from bronchopulmonary symptoms. As of this date (April 1954) there has been no change in postoperative status or chest roentgenogram (Figure 2B).

Gross examination of the resected pulmonary lobe revealed a 2.5 cm. nodule within the substance of the lung. Section of the nodule disclosed it to be fairly well circumscribed and encapsulated and slightly trabeculated with several focal dark reddish areas of discoloration. No connection existed between this lesion and the bronchovascular structures.

*By Joseph Goorwitch, M.D.

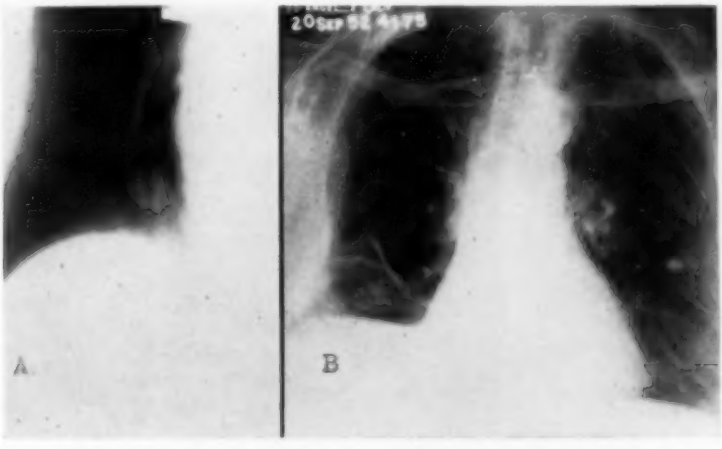

FIGURE 2A FIGURE 2B

Figure 2A: Planigram showing the "coin" lesion.—*Figure 2B:* Roentgenogram seven weeks postoperatively.

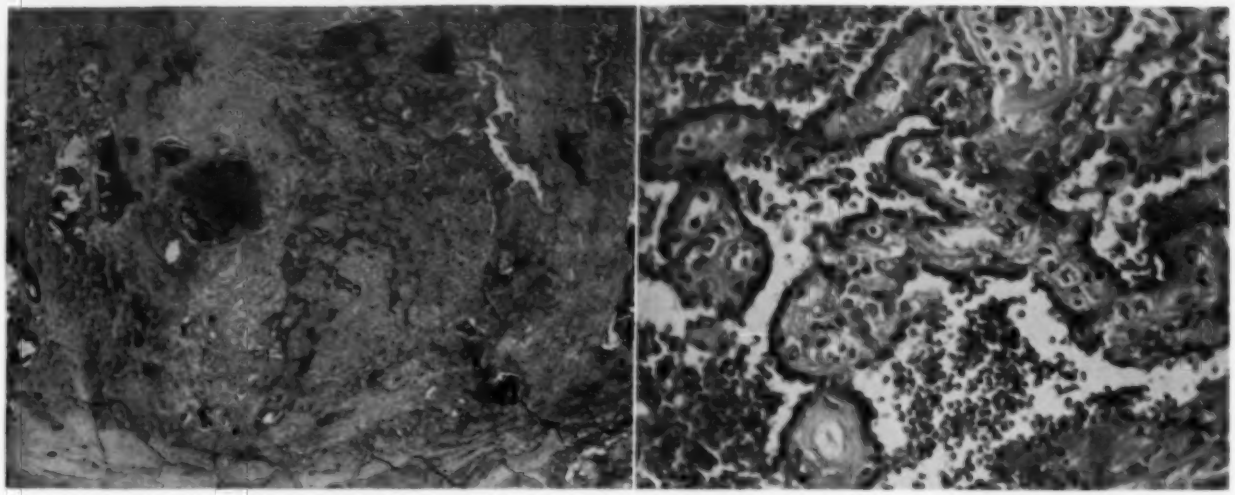

FIGURE 3 FIGURE 4

Figures 3 and 4: Low and high magnifications of sections of "coin" lesion showing capillary hemangioma.

TABLE I

Incidence of Bronchogenic Cancer and Pulmonary Capillary Hemangioma in Patients with Circumscribed Solitary Pulmonary Lesions.

Author and date	Pulmonary lesion described	Number of patients & method of diagnosis	Sex and age	Incidence of	
				Bronchogenic cancer	Capillary hemangioma
Thorton, Adams & Bloch 1944	Solitary circumscribed tumor	23 with & without operation	?	12 (52%)	1*
Effler, Blades & Marks 1948	Asymptomatic Solitary peripheral mass	24 all operated	Males 19-57 Av. 35	1 (4%)	0
O'Brien, Tuttle & Ferkaney 1948	"Coin" lesions	21 all operated	?	8 (38%)	0
Johnson, Clagett & Good 1949	Peripheral circumscribed mass	53 all operated	?	35 (66%)	0
Mahon & Forsee 1950	Round peripheral lesion	55 all operated	?	2 (3½%)	0
Sharp & Kinsella 1950	Asymptomatic isolated nodule 1-4 cm. in dia.	55	?	12 (22%)	0
Harrington 1951	Asymptomatic circumscribed lesions	16 all operated	?	2 (13%)	0
Abbott, Hopkins, Leigh and Van Fleit 1951	Solitary peripheral mass larger than 1 cm. in dia.	81 all operated	?	31 (38%)	0
Effler 1951	Asymptomatic solitary tumor	16 all operated	28-66 Av. 50	6 (37%)	0
Fink 1951	Solitary nonhilar lesion up to 6 cm.	30 with & without operation	?	10 (33%)	0
Abels & Ehrlich 1951	Asymptomatic single circumscribed density	21 with & without operation	?	5 (24%)	0
Condon 1952	Asymptomatic round solitary lesion	27 all operated	50-	17 (63%)	1**
Hood, Good, Clagett & McDonald 1953	Solitary circumscribed lesion	156 all operated	M 57% F 43% 6-69	25 (16%)	1
May, Rose and Dugan 1954	Solitary lesion on routine film	36 with & without operation	Males 21-70	8 (22%)	0

*Not described as capillary; excised by pneumonectomy.

**Not described as capillary; method of excision not stated.

Microscopic examination (Figures 3 and 4) of multiple sections thru the tumor nodule revealed an essentially similar appearance. The tumor is apparently well encapsulated and demarcated from the adjacent pulmonary tissue. There are foci of round cell infiltration and hemorrhage in the capsule. The tumor itself, in places, is made up of broad strands of somewhat oval to polyhedral cells having a generally uniform appearance and interspersed with scattered leucocytes. In most areas of the tumor, however, there is a papillary proliferation resulting in many vascular spaces lined by endothelial cells covering the papillary projections. The stroma in many of these proliferations has an almost hyaline appearance. A few small multi-nucleated cells are scattered among the others. The pulmonary parenchyma and the bronchial tree are not remarkable.

While there is some variability in the histological appearance of the tumor from somewhat solid to more papillary vesicular areas it is thought that, in view of the gross and microscopic appearance with complete encapsulation, this is a benign tumor showing moderately active intrinsic proliferation. Diagnosis: capillary hemangioma of the lung.*

Discussion

Due to the extremely low apparent incidence of capillary hemangioma in the lung as well as to its benign nature, it obviously does not present a serious diagnostic or therapeutic problem; however, for the sake of completeness of the differential diagnosis of pulmonary "coin" lesions, capillary hemangioma must be included along with other benign though more common lesions.

The increasing popularity enjoyed by chest roentgenography in modern times is resulting in the discovery of more instances of "coin" lesions. Due to the lack of uniformity in what constitutes a pulmonary "coin" lesion, an isolated pulmonary nodule, a solitary peripheral pulmonary mass or a peripheral circumscribed pulmonary tumor, it is difficult to compare statistical data found in the literature.[5, 18] However, because these same sources (Table I) reveal that the incidence of primary lung carcinoma in patients with solitary spherical intrapulmonary non-hilar masses can be as high as 66 per cent depending on sex and age, it becomes essential to determine the nature of such lesions as soon as discovered. As a more aggressive attitude toward these lesions of unknown nature becomes prevalent more inflammatory as well as benign neoplastic lesions will be resected with possible discovery that the lesion which constitutes the subject of this report is perhaps not as rare as it appeared to be.

SUMMARY

This case of benign capillary hemangioma of the lung radiologically described as a solitary spherical intrapulmonary non-hilar density ("coin" lesion) was treated by excision because of inability to exclude neoplasm.

RESUMEN

Este caso de hemangioma capilar benigno descrito como una densidad solitaria, esférica intrapulmonar no hilar (lesión "en moneda"), se trató por la excisión a causa de imposibilidad de excluir la neoplasia.

RESUME

L'auteur présente un cas d'angiome capillaire du re poumon qui se présente radiologiquement comme une ombre arrondie intra-pulmonaire. L'exérèse en fut pratiquée, prce qu'il était impossible d'affirmer qu'il ne s'agissait pas d'un cancer.

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Primary Chondroma of the Lung*

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Reports in the literature are confusing in regard to differentiation between chondroma of the lung and hamartoma of the lung. According to Hochberg and Pernikoff[1], these tumors are distinct and different forms of neoplastic disease. Bragg and Levene[2] feel that in several articles in the literature dealing with pulmonary hamartoma these are incorrectly referred to as chondroma. The confusion dates from Albrecht (1904), when his differentiation of hamartoma as a benign mixed tumor occurring in various organs, including the lung, was made. According to him, hamartomata are not true tumors, but rather tumor-like mal-formations due to abnormal mixing or development of the normal components of that organ. The abnormality may take the form of a change in quality, arrangement, or degree of differentiation, or comprise variations of all three phases. Hochberg and Pernikoff consider chondroma of the lung a rare tumor, whereas in contrast, Bragg and Levene claim that hamartoma is not a rare tumor.

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Chondroma of the lung is described as variable in size, the smaller one being generally attached to a larger bronchus, and the larger tumors usually within the pulmonary parenchyma. Chondromas are round or ovoid, firm in consistency and covered by a semi-translucent capsule. Trabeculations pass from the capsule throughout the tumor substance, forming lobules. Accompanying changes in the surrounding lung tissue depend upon the location, size, and duration of the primary tumor. Histologically, these tumors simulate normal cartilage. The fibrous tissue capsule and trabeculation often are vascular. The core of the tumor may have calcification and bone formation. At other times there may be hemorrhage in the tumor, myxomatous degeneration, and sarcomatous changes.

However, the tumors described as pulmonary hamartoma occur as a solitary, discrete, lobulated, solid mass, often containing calcified areas situated in the parenchyma near the pleura, and surrounded by normal appearing lung. Cartilage, primitive connective tissue, and islands of glandular tissue are the usual constituents, with fat, bone, smooth muscle, lymphoid tissue and amorphous calcifications occurring less frequently. Although hamartoma contains cartilaginous elements, it is not comprised chiefly of that tissue, hence Moller (1933),[12] called these mixed tumors.

In the literature some 100 cases have been reported, at least 75% of which were seen at autopsy. Verga (1932) was able to collect 58 cases and personally found 2 cases in a series of 20,000 autopsies. Hochberg and Pernikoff observed 2 cases in their series of 4,800 autopsies, and adding cases from the literature, reported a total of 78 cases. McDonald et al[8] reported 20 instances of "hamartoma (often called chondroma)" of the lung, from 8,000 autopsies.

Although the tumor may occur in persons of all ages, the greatest incidences are after the fourth decade of life and there is a preponderance of three or four males to one female. The distribution corresponds roughly to the relative size of the different lobes, the right lower lobe being the most common, particularly in a subpleural location. The size varies from that of 1 cm. in diameter to one described as 20 x 16 x 9 cms. Virchow reported a case where three chondromata were present in one lung; one near the hilum, one within the parenchyma, and the third in the pleural region.

The case presentation has the microscopic picture of chondroma and lay in the periphery of the lung, right lower lobe. It has a known existence of 20 years.

Case Report

This is a 61 year old white male who suffers from Schizophrenic Reaction, Paranoid Type, Chronic Severe, manifested by seclusiveness, disinterestedness, severe feelings of hostility toward his surroundings, suspiciousness, and is uncooperative. He has been mentally ill for many years and has been a resident of a mental hospital for some 22 years. He is a veteran of World War I. The original evidence of pulmonary tumor was found in an x-ray film taken December 5th, 1930. Because his appetite had been poor and he was losing weight a suspicion of pulmonary tuberculosis was entertained. This film was reported as essentially negative except for what was described as "large calcified gland and adhesions to the diaphragm in the right lower lung field." No other pulmonary lesion was observed. X-ray films taken in 1935, 1937 and 1939 were similar with no evidence of pulmonary tuberculosis. In the last report of 1939, the right lung at the base, posteriorly, presented a definite oval increased density approximately 3 x 4 cm. At this time the radiologist felt that the density of the right base had been

present since his admission in 1930. A review of the films shows this density to stay in the same location, but to be somewhat larger than in 1930. It was believed this represented a cyst, or a benign tumor, possibly a fibroma. Subsequent to this he developed occasional respiratory complaints, which were transient and incidental, and subsequent x-ray films during the next 10 years showed no change in the well circumscribed density at the right base. In 1949 he had acute appendicitis and appendectomy. In the latter part of the same year he had acute cholecystitis and subsequent cholecystectomy. In the last 22 years a search was made for tubercle bacilli and for ecchinococcus cysts, which were never found.

In 1950 he was observed again for loss of weight and poor appetite. No cause for symptoms could be discovered except possibly for this mass in the chest, which was felt now to be undergoing degenerative changes. A surgical consultation made the diagnosis of hamartoma and suggested thoracotomy. On July 10, 1951, resection of the pulmonary tumor in the right lower lobe was performed through the bed of the eighth rib. The tumor was easily resected from the parenchyma and lay just beneath the visceral pleura.

Pathological examination described a grayish yellow mass measuring 5 x 3.8 x 2.8 cm. One aspect firmly attached to the pleura over an area of 4.5 x 4 cm. The pleura was thickened and opaque. The remainder of the tumor was partially covered by a thin capsule-like structure. It was moderately firm in consistency and one section was composed of lobules of various size. There were large pearly white and bluish gray areas as well as yellow zones of fat. The white and gray areas were firm, the latter having the consistency of cartilage. There was no apparent infiltration into or beyond the capsule, or into the attached pleura. Histological examination showed large areas composed of cartilaginous tissue. The cartilage cells showed variations of size as well as variation in the size of the lacunae. Different areas showed different amounts of matrix. In some a fine fibrillar connective tissue was seen. The cartilage tissue was present in the form of lobules with fairly well defined periphery. The surrounding tissue was chiefly fat. In addition, there were areas composed of myxoid tissue, with spindle and stellate cells, and a fine pale blue staining inter-cellular matrix, also showed inter-lacing fibers. Some portions of the tumor were lined by tall columnar, ciliated epithelium, with oval nuclei tending toward the base. In some areas a loose substantia propria, with a small number of plasma cells, was present beneath the epithelial lining. On one edge a small fragment of compressed pulmonary tissue was recognizable. The diagnosis was chondroma.

The patient made an uneventful post-operative recovery and is well, as far as his chest is concerned, 18 months after the operation.

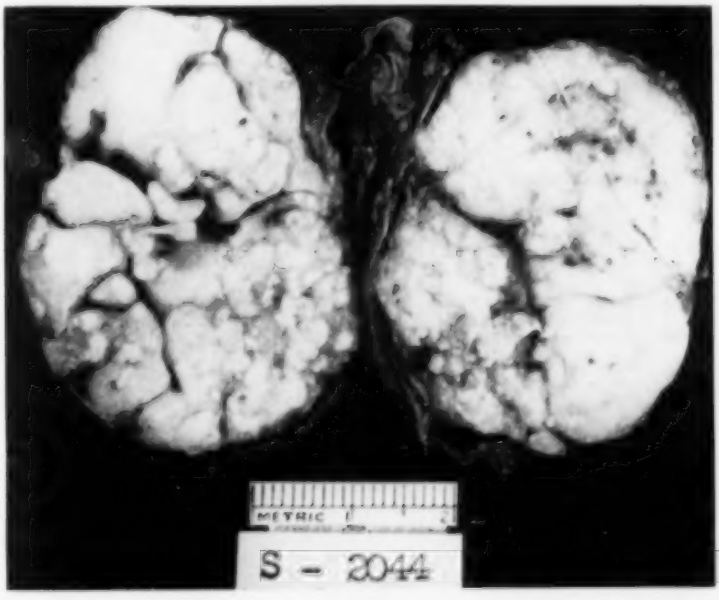

FIGURE 1: Primary Chondroma of the Lung Gross.

Comments

The microscopic findings in the present case conform mainly to the diagnosis of chondroma and yet clinically have the characteristics of hamartoma in that it lay in the periphery of the lung and was of long standing (at least 20 years known existence). This suggests that chondroma and hamartoma of the lung are quite similar, and the differentiation is not as distinct as has been described. To avoid further confusion it is suggested that these tumors should be considered the same, with one being a variation of the other.

The majority of cases of hamartoma of the lung have been classified as chondroma because of the predominance of cartilage. It is also probable that some cases in which the glandular elements predominate that a bronchial adenoma was diagnosed. McDonald et al[8] described three benign pulmonary tumors which were strikingly similar and unusual. They called them hamartoma (often called chondroma) and all the tissues found in these tumors correspond with those found normally in the bronchi, although lacking in orderly arrangement. These tumors were supposed to be the result of abnormal development of bronchial analage, therefore, called hamartoma, (failed, erred). There is a contention also that hamartoma may contain cartilaginous elements, but is not chiefly comprised of that tissue.

It is our surmise that the chondroma and the hamartoma as described in the literature may be one and the same tumor, with variations. This is a matter of histologic debate and it seems that the histogenesis is the

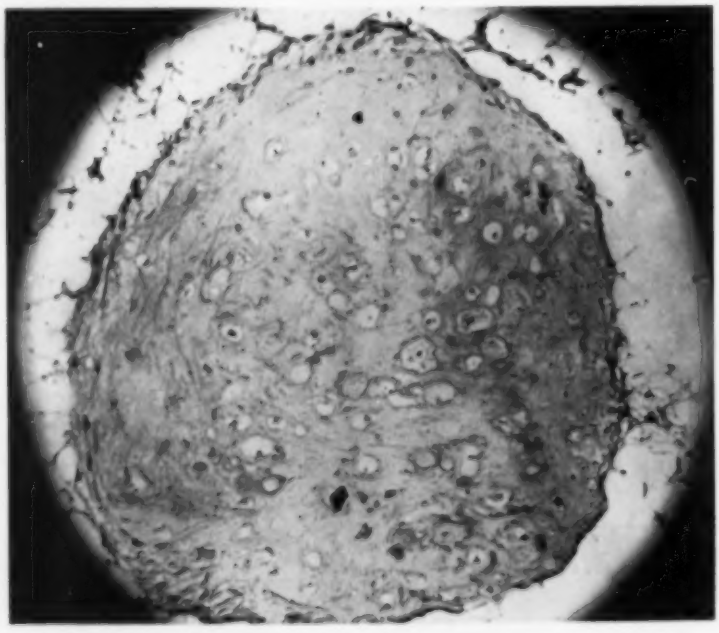

FIGURE 2: Primary Chondroma of the Lung Microscopic Section.

same. It is quite apparent that the chondroma and the hamartoma are alike clinically in that they occur in practically the same places and have the same slow growth and provoke few or no symptoms. It is believed that hamartoma is strictly a benign tumor and although there is a suspicion of chondroma undergoing sarcomatous changes, none have been reported. Again this makes one believe that the two tumors are alike. Both tumors are diagnosed roentgenologically and they should be suspected in every case of a solitary tumor of the lung. Frequently the diagnosis can only be made on microscopic examination. It is agreed that excision of the tumor is the recommended therapy. Until we are more adept at x-ray diagnosis excision is recommended because by necessity the tumor must be differentiated from bronchogenic carcinoma, a solitary metastasis, bronchial adenoma, tuberculoma and ecchinococcus cyst.

Two theories have been proposed as to the origin of these tumors of the lung. The first assumes that bronchial wall irritation will cause hyperplasia of cartilage and tumor formation. This theory would explain the origin of some of the tumors found within the bronchial wall. On the other hand, the second theory, which is more generally accepted explains tumor formation as the results of abnormal development of the bronchial analage, with alteration in the development of the cartilaginous elements. In the development of the lung the centers which form the cartilage in the mesoderm of the bronchus are scattered and the lung grows much more rapidly after the bronchial wall is developed. It is assumed that in the expansion of the lung after birth some displacement of these aberrent elements will later give rise to peripheral parenchymal tumors.

The symptoms induced by chondroma of the lung are dependent upon the size, location and the effects in the neighboring tissue, and as shown, many of these are entirely asymptomatic and only discovered at autopsy. The neoplasm that resides in the neighborhood of the main bronchus will provoke symptoms characteristic of bronchial irritation, such as asthmatic and dyspneic symptoms if encroaching on the lumen of the bronchus. The tumors in the periphery of the lung will remain asymptomatic until they cause erosion of a vessel, or local or referred pain when coming close to the chest wall. Since these tumors grow slowly without appreciable change in size as demonstrated in this case (20 years known duration) the symptoms will be minimal and transitory and probably will not interfere with peripheral pulmonary function. In some cases the tumor was first seen on x-ray film. The roentgen diagnosis is not always possible and rests mainly on the presence of calcification within the tumor. This usually is sharply defined in the lung parenchyma with clear lung tissue surrounding it. Some reports describe lobulation of the margins. The location is usually peripheral and subpleural.

SUMMARY

Primary chondroma of the lung is a benign tumor. One in the periphery of the right lower lobe of the lung with a known existence of 20 years with operation and recovery is presented. Chondroma and hamartoma as described in the literature may be one and the same tumor, with varia-

tions. This is a matter of histological debate and it seems that the histogenesis is the same. Both tumors are alike clinically in that they occur in practically the same places and have the same slow growth and provoke few or no symptoms. Excision is recommended until we become more adept at x-ray diagnosis.

RESUMEN

El condroma primario del pulmón es un tumor benigno. Se presenta uno situado en la perferia de lóbulo inferior derecho, cuya existencia se había conocido por 20 años, el cual fué operado y se recuperó. El condroma y el hamartoma, tal como se describen en la literatura, pueden ser el mismo tumor, con variantes.

Este es un motivo de discsión histológica y así parece respecto de la histogénesis. Ambos tumores se asemejan clínicamente en que aparecen prácticamente en los mismos lugares, tienen un crecimiento lento y provocan pocos síntomas o ningunos. Se recomienda la excisión, mientras no vengamos a hacer un diagnóstico radiológico más adecuado.

RESUME

Le chondrome primitif du poumon est une tumeur bénigne. L'auteur rapporte un cas qui siégeait à la corticalité du lobe inférieur du poumon droit, et dont l'existence était connue depuis 20 ans. Il fut opéré et suivi de guérison.

Le chondrome et l'hamartome tels qu'ils sont décrits dans la littérature sont peut-être une seule et même tumeur avec quelques différences. Il y a là un problème qui demande une discussion histologique et il semble bien que l'histogénèse soit commune. Les deux tumeurs sont tout à fait semblables au point de vue clinique. Pratiquement, elles surviennent au même point, elles ont le même développement lent, et le même silence symptomatique. Tant que nous ne sommes pas capables d'affirmer leur nature par un diagnostic radiologique exact, il faut en pratiquer l'extirpation.

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Supraventricular Tachycardia

Following Oral Administration of Banthine

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The ganglionic blocking effects of methantheline (banthine) bromide, a quaternary amine, have been applied therapeutically to a variety of clinical conditions. Since Longino and associates[1] demonstrated the inhibitory effects of banthine on gastric secretion and gastric motility, there have been favorable clinical results reported in the therapy of peptic ulcer,[2] hyperhidrosis,[3] pancreatitis[4] and enuresis.[5] In 1952, Fisher and Winsor[6] suggested the oral use of banthine as a replacement for atropine sulfate in the preoperative preparation of patients undergoing chest surgery. These observers noted an absence of tachycardia that is so frequently seen after parenteral administration of atropine sulfate. In 1953, Gregory and his co-workers[7] reported their experiences with ganglionic blocking agents administered to patients suffering attacks of bronchial asthma. An increase of vital capacity was measured in some patients following banthine.

The reported side-effects on the cardiovascular system have been varied and contradictory. Smith, Woodward, Janes and Dragstedt[8] observed no change in pulse rate when banthine was given orally in doses as high as 100 mg. every four hours. Winsor[9] states that no significant degree of tachycardia developed in patients given less than 75 mg. orally. Kern, Almy and Stolk[10] found an average increase in pulse rate of 17 beats per minute in most of their patients given 100 mg. of banthine. On the other hand, a temporary increase in pulse rate was observed after the intravenous administration of 100 mg. of banthine in practically every instance.[11] Longino[1] reported the average increased pulse rate of 20 beats per minute after intravenous administration of banthine.

It is the purpose of this communication to stress the varied effects of banthine on the cardiac rate and to record a case in which 50 mg. of banthine, orally, caused attacks of supraventricular tachycardia.

Case Report: This man of 50 years was first seen in 1950. He complained of indigestion and pressure around the heart. He stated that his heart, at times, "will beat as though it might fly out of the chest." There was no undue dyspnea on exertion. He complained of a sour taste after eructation. Belching would relieve the epigastric discomfort occurring one-half to one hour after meals. There was a history of the usual childhood diseases. In 1944, while in the Pacific area, he contracted malaria. No heart disease was found at this time following study for precordial distress. The family history was non-contributory.

Physical examination presented an introspective male who weighed 131 pounds and was 67 inches tall. The blood pressure was 120/80 mm Hg. The pulse was regular and the rate was 78 beats per minute. The thyroid gland was normal. The lungs were normal to ascultation and precussion. There were no rales after coughing. The aortic second sound had a tambour-like quality. No murmurs were heard. There was a point of tenderness to the right of the umbilicus on deep pressure and slight pararectus muscle spasm. The liver and spleen were not palpable.

Urinalysis and blood count were normal. Fluoroscopy of the chest was essentially negative. A chest x-ray film disclosed a minimal stabilized tubreculous lesion in the right supraclavicular area. The heart was normal in size and contour. The electrocardiogram showed a tendency for the S-T segments in L2 to be "saggy." Gastric

analysis showed hyperacidity. Blood Wassermann reaction was negative. A sputum specimen was negative for tubercle bacilli. The PPD skin test was negative to the first strength.

Dietary precautions with vitamin supplements brought relief of symptoms until January, 1953. Recurrence of symptoms prompted repetition of studies. An acute duodenal ulcer was found on x-ray study. Frequent auricular ectopic beats, some showing retrograde conduction, appeared in the electrocardiogram. A bland diet, anti-acid powder, mild sedative and vitamin supplement were prescribed. The auricular arrhythmia responded nicely to pronestyl. In June, 1953, due to exacerbation of ulcer syndrome, 50 mg. of banthine every 12 hours was added to the therapeutic regime. Shortly, thereafter, he experienced sudden attacks of substernal pressure associated with marked dyspnea precipitated by flushing of the face, and followed by a feeling that "my heart would fly out of my chest." His wife, a registered nurse, found his pulse rate to be 134 and 136 beats per minute in two of these attacks. They would last for two or three hours. Following the third attack, it was realized that these episodes occurred about 30 to 40 minutes after taking 50 mg. of banthine. He voluntarily discontinued this medication and experienced no recurrence.

Due to the unusual reaction reported, he was asked to come in for study. The Master two-step test showed no evidence of coronary insufficiency. The exercise tracing did show infrequent auricular ectopic beats. After a period of time, he was given 50 mg. of banthine and told to wait in the recovery room. Thirty-two minutes later, he summoned the nurse and stated that he suddenly felt hot and had a tightness in the chest—that he felt like he did after taking a banthine tablet. He was pallid and the pulse was 130 beats per minute with regular rhythm. The blood pressure was not taken. Figure 1 shows lead II of the electrocardiogram, resting and after banthine. Because of ill effect experienced, he has refused to take smaller doses of this compound.

Discussion

Banthine appears to have a ganglionic blocking action on the autonomic nervous system with an additive parasympatholytic effect at the myoneural junction.[6, 9] The use of banthine in place of atropine in the preparation of patients for chest surgery has been suggested by Fisher and Winsor.[10] They observed that the oral administration of 100 mg. of banthine one hour before surgery would frequently abolish the rapid heart

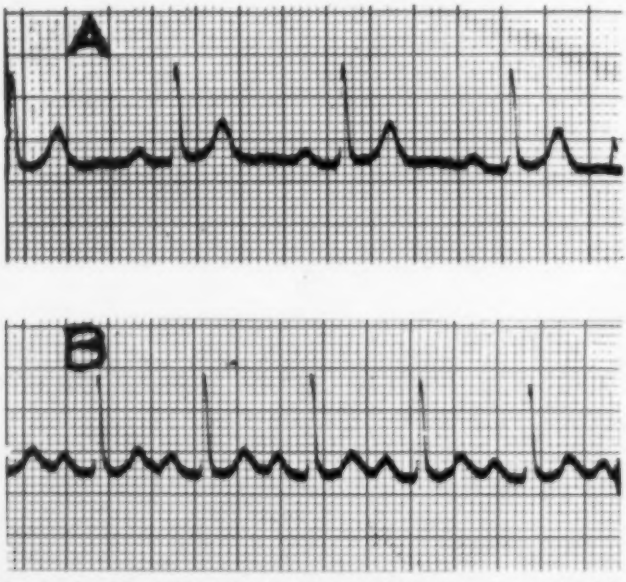

Figure 1: A, standard lead II of tracing before Banthine. *B*, standard lead II taken approximately 40 minutes after 50 mg. of Banthine was given orally showing supraventricular tachycardia.

rate often seen after atropine. They believed this favorable effect due to blockage of the vagal impulse at the myoneural junction and a depressing action of the sympathetic ganglions. In view of the varying reports concerning transient tachycardia following oral administration of the same dosage of banthine, this quaternary amine must inconsistently depress the sympathetic nervous system. The experience in this case is evidence of this. The disturbances on the cardiovascular system in this case suggests that a test dose of banthine be administered routinely to detect any untoward effect before this compound is used in therapy.

SUMMARY AND CONCLUSIONS

Attacks of supraventricular tachycardia occurring about 30 to 40 minutes after oral administration of 50 mg. of banthine are reported. The tachycardia persisted for two to three hours. The routine administration of a test dose is advised when banthine is contemplated in the therapeutic regime.

SUMARIO Y CONCLUSIONES

Se reportan ataques de taquicardia supraventricular que ocurrieron cerca de 30 a 40 minutos después de la administración oral de 50 mg. de bantine. La taquicardia persistió por dos a tres horas. He recomienda la administración rutinaria, de una dosis de prueba cuando-se considera un régimen terapéutico con bantine.

RESUME

L'auteur rapporte un cas d'attaque de tachycardie supraventriculaire, survenue environ 30 ou 40 minutes après l'administration buccale de 50 mmg. de banthine. La tachycardie persista pendant deux ou trois heures. L'auteur conseille l'administration préalable d'une dose d'essai lorsque la banthine doit faire partie du traitement projeté.

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Editorial

Pulmonary Emphysema Defined

The term, pulmonary emphysema, has been incorrectly assumed, and is frequently diagnosed incorrectly by the clinician, the roentgenologist, and the pathologist. In 1944, Christie[1] stated the term emphysema has been so loosely applied that much confusion both in *diagnosis and therapy* has resulted. In fact, there are few clinicians and pathologists who agree on a definition of pulmonary emphysema. Under etiology, Christie[2] in 1954 states, "Numerous hypotheses have been advanced to explain the cause of this type of emphysema but most are based only on conjecture and are incapable of experimental proof or analysis." Frequently hypertrophy of a part or the whole lung is considered to be emphysema, even with the occurrence of better function. For a long time hypertrophy of lung tissue was believed to eventually change into an emphysematous lung. There is proof now that such changes do not occur and that a hypertrophied lung may function more than a normal lung (Ornstein[3]). What is the reason for all this confusion? When dyspnea is constant and the chief symptom, emphysema of the lung is tagged on the diagnosis no matter what may be the cause of the dyspnea. There should be a careful search for the cause of the labored breathing. Though there are many causes for dyspnea, two factors should be determined. One, a frequent cause of dyspnea, the 'ventilatory reserve.' This can easily be determined by dividing the 'resting minute ventilation' into the maximum minute breathing capacity. The liters of resting minute ventilation and the amount of liters of air that can be forced into the lung in that minute will give a good estimate of the ventilation of the lung. When the factor is six or below the patient is dyspneic. This was proved by reducing the vital capacity in a group of normal males who had healthy lungs and were not dyspneic, and who became so when their normal vital capacity was reduced from 25 to 50 per cent of the normal by compressing in a canvas jacket the thorax and abdomen (Ornstein, Herman Friedman and Friedlander[4]). The explanation of this form of dyspnea is probably associated with changes in the wall of the thorax and the causes have not been solved at the present moment. This form of dyspnea has often been called emphysema, and still the lungs demonstrated good diffusion of oxygen and carbon dioxide. It is known that in emphysema of the lungs there is an atrophy of the stretched hemo-respiratory surfaces of the pulmonary alveoli and good diffusion would not occur.

Roentgenologists have called hypertrophied lungs emphysematous. Ornstein[3] has demonstrated such cases where the diffusion is within the normal range or below.

Pathologists are not too clear in differentiating hypertrophy from emphysema (Hartcroft,[5] Wells and Merriman,[6] and Wilson[7]).

Clinicians are apt to tag on the term of emphysema to any chronic pulmonary or heart disease when dyspnea is a constant symptom.

A definition of pulmonary emphysema should be used when there is marked interference with the diffusion of oxygen and carbon dioxide. Before associating dyspnea with pulmonary emphysema the diffusion of oxygen and carbon dioxide should be determined. This can be done after an oxygen debt is produced by estimating the percentage of oxygen that has been used up in the rebreathing bag (Ornstein et al.[4]) or by the po_2 in the femoral artery after using pulleys to produce an oxygen debt while breathing 15 per cent oxygen (Ornstein, Meyers and Eckman[8]). If the diffusion is not good, a diagnosis of emphysema can be made.

It is necessary to have a definition for emphysema so that methods of therapy may be considered for its cure. Many procedures have been used for the treatment of dyspnea and have been confused as therapy for emphysema.

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Teaching Chest Disease*
The Coin Lesion

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The "coin lesion" can be defined as any lesion which, on x-ray film of the chest, presents a peripheral well circumscribed shadow. It has been a difficult problem for the clinician because such a lesion may represent the most malignant or the most benign type of disease.[1]

In presenting this problem to a group of students, the following technique has been used successfully. First, x-ray films portraying a typical coin lesion are demonstrated. There may be six or eight films all showing a similar lesion. The students are asked to write down their diagnoses and then one film is picked from the group as an example. One will usually find that there are several diagnoses from the group for any one particular lesion. The student is asked to justify his diagnosis, and it soon becomes apparent that this justification can apply to any of the other lesions presented and that another student can equally well justify a different diagnosis. In short, the students now realize that it is impossible to differentiate one coin lesion from another.

They are then asked to list the various disease processes which could cause such an x-ray shadow. Such a typical list includes:

Bronchogenic carcinoma
So-called bronchial adenoma
Tuberculoma
Hamartoma
Inspissated tuberculous cavity
Coccidioidomycosis
Histoplasmosis
Abscess
Hemangioma
Arteriovenous fistula
Encapsulated effusion
Mesiothelioma
Encapsulated foreign body
Bronchogenic cyst
Other rare tumors (lipoma, fibroma, leiomyoma)
Sarcoma of the lung (rare)
Metastatic tumor

Having developed such a list, the prognostic implications and, therefore, the surgical considerations become obvious to the group. With the exception of a true tuberculoma and similar forms of fungus disease, all are most adequately handled surgically.

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It has already been agreed upon by the group that with the demonstrated x-ray film any of the listed diseases is a possibility.[2] Now, what are the further examinations which will help us in our differential diagnoses? Bronchoscopy will very seldom be of any value because the lesion is peripheral and, therefore, beyond the area which may be visualized with the bronchoscope. Sputum examinations may be of some value if positive either cytologically or bacteriologically. They are of no value if negative. Skin tests for tuberculosis and fungus disease are usually helpful if negative, but a positive reaction does not necessarily indicate the pathogenesis of the lesion. Further x-ray examinations will usually only serve to confirm the presence of a peripheral lesion, though lamination or focal calcification may be demonstrated.

The next question for discussion is the management of such a lesion. Is it safe to follow such a lesion with serial x-ray film examinations? One glance at the list the group has prepared will lead immediately to the realization that such a program is not safe in that malignancy is certainly a possibility. It, therefore, follows that the wise course to follow is thoracotomy with excision and immediate pathological examination.[3, 4] The further course of therapy can then be established on a firm basis.

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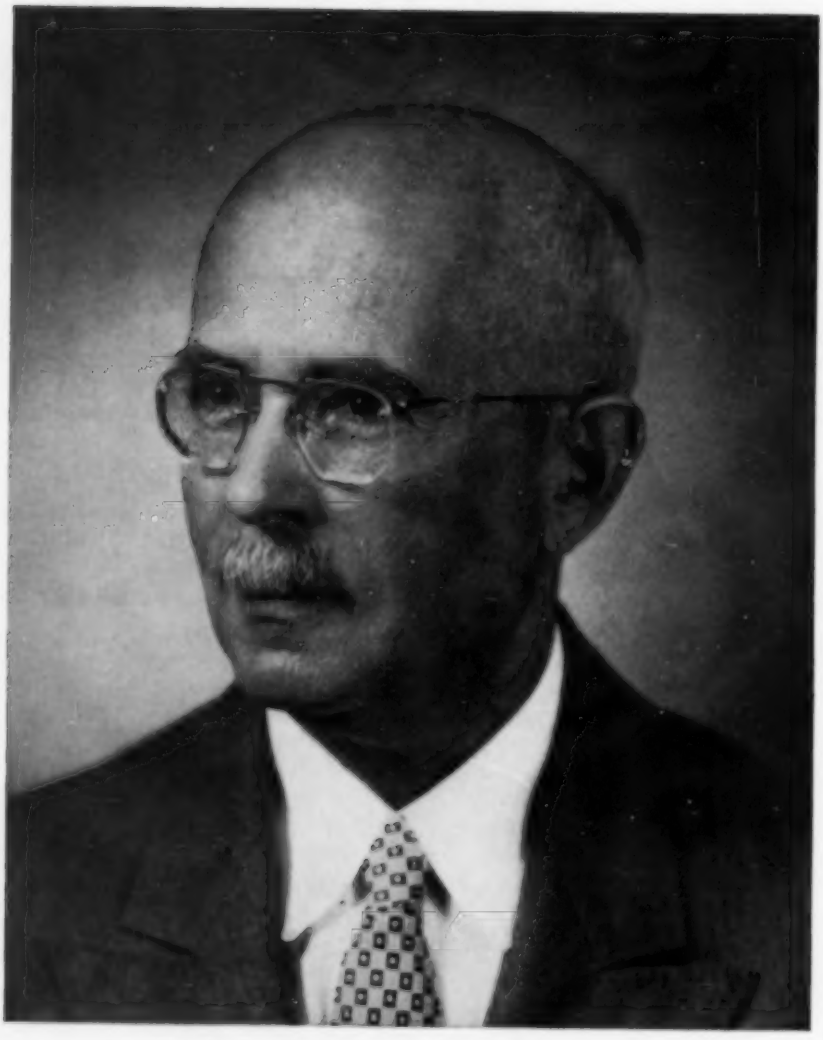

Andrew L. Banyai

PRESENTATION OF THE COLLEGE MEDAL FOR 1955 TO DR. ANDREW L. BANYAI*

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I am very grateful for the opportunity of speaking for the American College of Chest Physicians on this occasion. I have no illusions about being able to do justice to the subject of our attention this evening. But no member of this society needs to be reminded of the professional achievements of Dr. Andrew Banyai, and few of us need to be told of his personal qualities. A roll call of his personal friends would encompass the whole College of Chest Physicians.

He graduated from the Medical School of the University of Budapest in 1915. Thus, he was trained in the very center of that great European medical movement which had as its object nothing less than the transformation of the old art of medicine into a science. It is no exaggeration to say that Dr. Banyai has devoted his life to this great ideal of his medical school, the ideal of a humane medicine which would yet be a true science. One aspect of his dedication is his truly encyclopedic knowledge which can still startle even those who know him best by its remarkable depth and breadth. His abundance of published research has been a product of this same dedication. From the beginning, Dr. Banyai's publications were marked by a concern for accuracy and for detail—in short, a passion for truth—which make them models, not only for their own time, but for today. In the 1920's, when many students of tuberculosis were still searching for the universal panacea, Dr. Banyai's sense of complexity which any biological science must display, kept him working on fundamentals. As a consequence, he has been consistently on the frontiers of medical knowledge, and his work is of permanent value. His pioneer research in the development of formulae for estimating vital capacity by roentgenologic studies, his interest in the mechanism of cough and in expectorants, and his many articles on the subject of occupational therapy are typical of the basic contributions he has made in the last thirty years. But perhaps the greatest testimony to the real solidity of his research are his writings on pneumoperitoneum. Published in the early 1930's his studies have accomplished the feat of remaining the basic authority in its field in spite of being recorded a good ten years in advance of general application of the subject. In a period in which medical knowledge has been accumulated so rapidly, only research of the highest quality could maintain such authority.

But these achievements which would have been sufficient for any unusual career by no means exhausted Dr. Banyai's energy. He had a passion for medical science, but he also had a passion for making it prevail. I do not know whether Dr. Banyai has spoken in every state in the union or not, and I doubt very much that he would know. He did recollect speaking engagements in numerous other countries when I asked him, and I am sure that if the guests here tonight were to put together their recollections of the American meetings that they attended at which Dr. Banyai spoke, it would come to something very near to all the 48 states. He has been very much interested in the postgraduate program and has made himself available to postgraduate speakers' committees beyond the endurance of most of us. A large share of the credit for the success of the International Congress on Diseases of the Chest in Rome, Rio de Janeiro and most recently in Barcelona, is his, since he was responsible for the scientific programs of those meetings. He cheerfully undertook the difficult task of editor of a major contribution to the practice of medicine, the recent *Non-Tuberculous Diseases of the Chest*. As editor or advisor, he is connected with three major medical journals, and of course he has recently served as president of the American College of Chest Physicians.

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*Presented at the Presidents' Banquet, 21st Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 4, 1955.

**Chairman, Committee on Awards.

This distinguished career demands not only our respect but also our admiration. It is quite enough to account for the College medal we present tonight. But the fact is, we do not merely respect and admire Andrew Banyai; I know of no one in the medical profession who is more entirely loved by his colleagues. Somehow, in the spare moments of this remarkable career, he has found the time to become a great human being. It is not necessary that this observation be enlarged with adjectives—that I speak of his unfailing good humor, his gentleness, his sympathy and courtesy. The most important thing to point out that Dr. Banyai has apparently never felt himself to be other than a servant of truth and mankind. This is the reason, I think, that whenever he is discussed by his colleagues—and he is discussed by his colleagues a good deal—the qualities that finally agreed to best typify him are simplicity and humility. Simplicity, humility and in addition a dedication to truth—these are the qualities of Andrew Banyai, a truly great physician.

BIOGRAPHICAL DATA

Andrew L. Banyai was born January 19, 1893. He graduated from the Medical School of the Royal Hungarian University of Budapest with the degree of Doctor of Medicine in 1915. He began his training in diseases of the chest shortly thereafter. Since then he has carried on his professional activities as an internist, in private practice, in sanatorium and dispensary work. He became Clinical Director of Muirdale Sanatorium, Milwaukee, Wisconsin, in 1928. At the same time, he joined the Medical Faculty of Marquette University, Milwaukee, Wisconsin, where at the present time he is Associate Clinical Professor of Medicine.

Dr. Banyai is a Fellow of the American College of Chest Physicians, the American College of Physicians, a Diplomate of the American Board of Internal Medicine and was a member of the National Faculty of War-Time Graduate Meetings.

He started clinical research work in chest diseases during his early institutional affiliation. Since 1917 he has published more than one hundred papers in medical journals in this country and abroad.

Dr. Banyai is a member of the Editorial Board of *Diseases of the Chest*. He is also a member of the Editorial Advisory Committee of the *American Journal of Occupational Therapy* and of the Editorial Advisory Board of *GP*, the official journal of the American Academy of General Practice. Dr. Banyai serves as Chairman of the Council on International Affairs of the College and was instrumental in arranging the scientific programs for the First International Congress on Diseases of the Chest held in Rome, Italy in 1950, for the Second International Congress on Diseases of the Chest, held in Rio de Janeiro, Brazil in 1952 and for the Third International Congress on Diseases of the Chest held in Barcelona, Spain in 1954.

He served as President of the American College of Chest Physicians, 1952-1953, and has served in various other capacities steadfastly, with unselfish loyalty and devotion for many years.

ANNUAL MEETING AND ELECTION OF OFFICERS

The 21st Annual Meeting of the American College of Chest Physicians was held at the Ambassador Hotel, Atlantic City, New Jersey, June 1 through 5. More than 1,400 members and guests registered for the meeting, of which approximately 1,050 were physicians. Twenty-six technical exhibits were on display throughout the meeting. At the Annual Presidents' Banquet, held on Saturday, June 4, 750 physicians and guests were present to fill the main dining room and an adjoining room. Fellowship in the College was conferred upon 251 physicians at the Convocation ceremony which immediately preceded the Banquet. This was the largest Convocation Class in the history of the College.

The following Officers, Regents, and Governors were elected:

OFFICERS

President ..... James H. Stygall, Indianapolis, Indiana
President-Elect ..... Herman J. Moersch, Rochester, Minnesota
First Vice-President ..... Burgess L. Gordon, Philadelphia, Pennsylvania
Second Vice-President ..... Donald R. McKay, Buffalo, New York
Treasurer ..... Charles K. Petter, Waukegan, Illinois
Assistant Treasurer ..... Albert H. Andrews, Chicago, Illinois

REGENTS

District No. 2 ..... Edgar Mayer, New York, New York
District No. 4 ..... Dean B. Cole, Richmond, Virginia
District No. 6 ..... Willard B. Howes, Detroit, Michigan
District No. 10 ..... John F. Briggs, St. Paul, Minnesota
District No. 14 ..... James M. Odell, The Dallas, Oregon
District No. 15 ..... Jaime F. Pou, Hato Rey, Puerto Rico
District No. 16 ..... Hastings D. Walker, Honolulu, Hawaii
England ..... Richard R. Trail, London (to complete unexpired term of Sir Alexander Fleming, deceased)
Historian ..... Carl C. Aven, Atlanta, Georgia

GOVERNORS

Delaware ..... Gerald L. Beatty, Wilmington
Indiana ..... Jerome V. Pace, Rockville
Iowa ..... William H. Spear, Oakdale
Kentucky ..... T. Ashby Woodson, Louisville
Massachusetts ..... Howard Root, Boston
Montana ..... Frank I. Terrill, Deer Lodge
Nebraska ..... Max Fleishman, Omaha
Nevada ..... Robert C. Locke, Reno
New Hampshire ..... Francis J. Kasheta, Glencliff
New Mexico ..... Joseph E. J. Harris, Albuquerque
Ohio ..... David W. Heusinkveld, Cincinnati
Oklahoma ..... Donald W. McCauley, Okmulgee
Oregon ..... William S. Conklin, Portland
Rhode Island ..... Frank A. Merlino, Providence
South Carolina ..... J. Gordon Seastrunk, Columbia
South Dakota ..... William L. Meyer, Sanator
Wisconsin ..... Alfred A. Busse, Jefferson
Wyoming ..... Russell H. Kanable, Basin
Cuba ..... Francisco J. Menendez, Havana
Greater London ..... Geoffrey Bourne, London

College Chapter News

PENNSYLVANIA CHAPTER

The Pennsylvania Chapter will meet at the William Penn Hotel, Pittsburgh, September 21. The following program will be presented:

"Use of BCG Vaccine in the Control of Tuberculosis"
Anne B. Wagner, Pittsburgh

"Sarcoidosis—Diagnosis and Management"
Harold L. Israel and Maurice Sones, Philadelphia

"Pulmonary Enemies 1, 2, and 3"
Richard H. Overholt, Boston

A dinner and banquet will follow the scientific session, at which Dr. James H. Stygall, Indianapolis, Indiana, President of the College, and Dr. J. Winthrop Peabody, Sr., Washington, D. C., Chairman of the Council on Postgraduate Medical Education of the College, will be guest speakers.

NORTH INDIA CHAPTER

The North India Chapter met in Amritsar, February 18. Dr. Santokh Singh Anand presented two cardiac cases and a lobectomy case, which were discussed following his presentation. Dr. K. L.. Wig, Governor for North India, presented a series of x-rays, followed by discussion, and an exhibit of excised lung specimens was presented by Dr. Anand and Dr. Yadveer Sachdeva.

NEW CHAPTER OFFICERS

ALABAMA CHAPTER

President ..... James C. Nash, Decatur
Vice-President ..... Vivian H. Hill, Mobile
Secretary-Treasurer ..... Kellie N. Joseph, Birmingham

ARIZONA CHAPTER

President ..... Leslie B. Smith, Phoenix
Vice-President ..... Hans F. Stein, Tucson
Secretary-Treasurer ..... B. L. Snyder, Phoenix

CALIFORNIA CHAPTER

President ..... Walter B. Brown, Livermore
Vice-President ..... Marvin S. Harris, Los Angeles
Secretary-Treasurer ..... Mortimer Benioff, San Francisco

EAST INDIA CHAPTER

President ..... P. K. Sen, Calcutta
Vice-President ..... K. N. De, Calcutta
Secretary-Treasurer ..... P. K. Ghosh, Calcutta

FLORIDA CHAPTER

President ..... Hawley H. Seiler, Tampa
Vice-President ..... Jack Reiss, Coral Gables
Secretary-Treasurer ..... William L. Potts, Lantana
Program Chairman ..... Clarence M. Sharp, Jacksonville

GEORGIA CHAPTER

President ..... Clarence Mills, Atlanta
Vice-President ..... Fred Crenshaw, Rome
Secretary-Treasurer ..... F. Levering Neely, Atlanta
Program Chairman ..... Carl C. Aven, Atlanta

ILLINOIS CHAPTER

President ..... Albert H. Andrews, Chicago
Vice-President ..... John A. Mathis, Pinckneyville
Secretary-Treasurer ..... Gordon L. Snider, Chicago

KANSAS CHAPTER

President ..... Carl J. Wilen, Manhattan
Vice-President ..... Martin J. FitzPatrick, Kansas City
Secretary-Treasurer ..... Paul H. Wedin, Wichita
Program Chairman ..... John L. Morgan, Emporia

MEXICAN CHAPTER

President ..... Fernando Rebora, Mexico City
Vice-President ..... Enrique Staines, Mexico City
Secretary-Treasurer ..... Rafael Ibarra, Mexico City

MISSISSIPPI CHAPTER

President ..... Lee R. Reid, Jackson
Vice-President ..... H. Karl Strauss, Jackson
Secretary-Treasurer ..... Helen C. Bernfield, Jackson (re-elected)

MISSOURI CHAPTER

President ..... Bernard Friedman, Koch
Vice-President ..... John W. Polk, Mount Vernon
Secretary-Treasurer ..... David Nafe Kerr, St. Louis (re-elected)

NEW ENGLAND STATES CHAPTER

President ..... John B. Andosca, Boston, Massachusetts
Vice-President ..... Frank G. Seldon, Manchester, New Hampshire
Secretary-Treasurer ..... Joseph N. Corsello, Providence, Rhode Island

NEW JERSEY CHAPTER

President ..... Samuel Cohen, Jersey City
President-Elect ..... Paul Geary, Plainfield
Vice-President ..... Wilbur F. Jehl, Verona
Secretary-Treasurer ..... Lewis F. Baum, South Orange

NEW YORK CHAPTER

President ..... Mark H. Williams, Binghamton
1st Vice-President ..... Ida Levine, Brooklyn
2nd Vice-President ..... Francis W. O'Donnell, Buffalo
Secretary-Treasurer ..... Harry Golembe, Liberty (re-elected)

OHIO CHAPTER

President ..... Howard S. Van Ordstrand, Cleveland
Vice-President ..... Harold L. Humphrey, Columbus
Secretary-Treasurer ..... Francis G. Kravec, Youngstown (re-elected)

OKLAHOMA CHAPTER

President ..... Forrest P. Baker, Talihina
Vice-President ..... William G. Crandall, Sulphur
Secretary-Treasurer ..... George L. Winn, Oklahoma City (re-elected)

PERU CHAPTER

President ..... Ramon Vargas Machuca, Lima
Vice-President ..... Luis Angel Morales, Lima
Secretary ..... Victor Narvaez O., Lima
Treasurer ..... Alejandro Flores Degregori, Lima

TENNESSEE CHAPTER

President ..... Robert McCracken, Nashville
Secretary-Treasurer ..... Sheldon E. Domm, Knoxville
Vice-President ..... P. M. Huggin, Knoxville (re-elected)

WISCONSIN CHAPTER

President ..... Karl E. Kassowitz, Milwaukee
Vice-President ..... Philip P. Feingold, Milwuakee
Secretary-Treasurer ..... Leon H. Hirsh, Milwaukee (re-elected)

CARDIOVASCULAR RESEARCH AWARD

As announced in the April issue of DISEASES OF THE CHEST, the Committee on Cardiovascular Diseases of the Council on Research has offered an award of $500 for the best manuscript on "Acute Pulmonary Edema."

The study may be of either an experimental or a clinical type and may include problems of therapy. The original work, based on personal research, should be presented before May 1, 1956. It may consist of an unpublished manuscript or a recently published article (after April 1, 1955).

If the manuscript is unpublished, publication may take place either in *DISEASES OF THE CHEST* or in another journal according to the wish of the author.

For further information, please communicate with Dr. Aldo A. Luisada, Chairman, Section on Cardiovascular Physiology, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

ANNOUNCEMENT OF RESEARCH FUNDS

Applications for support of investigations on bronchiectasis, bronchial asthma, pulmonary emphysema and fibrosis, insofar as these diseases are affected by the common cold, may now be sent to the Council on Research of the American College of Chest Physicians by submitting an appropriate budget, as well as an abstract of not more than 250 words.

The proposed research, if recommended by the Council on Research, will then be submitted to the Scientific Advisory Committee of the Common Cold Foundation. Since the Foundation has recently expressed its interest in studies on the complications of the common cold as they affect these clinical entities, members of the College who are interested in obtaining funds for such studies are urged to send in their applications promptly. Please address Council on Research, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY

The Fourth International Congress of Bronchoesophagology will take place in Buenos Aires, Argentina, October 28 and 29, 1955. The President of the Congress will be Dr. Antonio Carrascosa; the Vice-Presidents, Dr. Plinio de Mattos Barretto, Dr. Anibal Grez and Dr. Julio C. Barani; and the General Secretary, Dr. Juan Carlos Arauz.

According to the preliminary program, there will be one session dedicated to diseases of the larynx, trachea and bronchi in children; and one devoted to non-cancerous esophageal disease. In addition, there will be a third session for papers on various subjects.

It is announced that the Fifth International Congress of Bronchoesophagology will be held in Vienna, Austria, in early September, 1956, following the Fourth International Congress on Diseases of the Chest taking place in Cologne, Germany, August 19-23, 1956.

For further information concerning these Congresses, please address Dr. Chevalier L. Jackson, Secretary, International Bronchoesophagological Society, 3401 North Broad Street, Philadelphia, Pennsylvania, U.S.A.

MEDICAL SERVICE BUREAU

Position Wanted

Principal public health physician, 46 years of age, seeks clinical or public health position in line with qualifications and experience; well qualified as organizer and supervisor of tuberculosis control program with parallel clinical and administrative experience in chest diseases and tuberculosis as director of chest clinics and state tuberculosis sanatoria; 22 years of continuous experience; highly recommended by physicians of international repute. Prefer New York, New Jersey, Ohio, California. Please give full details in reply. Address all inquiries to Box 293B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Positions Available

Staff physician wanted for a modern, well equipped and staffed, 210-bed tuberculosis hospital located in an attractive mid-western city of 180,000. Applicant must be sober, industrious, and qualified for Michigan license. Salary commensurate with qualifications and experience. Please address all inquiries to Box 280A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Assistant medical director wanted for 100 bed tuberculosis hospital. North American graduate; salary $8,500, complete maintenance. Apply Medical Director and Superintendent, District Five Tuberculosis Hospital, London, Kentucky, or State Tuberculosis Hospital Commission, New State Office Building, Frankfort, Kentucky.

Assistant medical director wanted to assist medical director in carrying out clinical program in chest and tuberculosis hospital. Salary $7,500-$9,000 depending on qualifications and experience. Must be eligible for Colorado licensure. Medical school faculty appointment probable. Wonderful opportunity for right person. Write S. H. Dressler, M.D., National Jewish Hospital, Denver, Colorado.

CALENDAR OF EVENTS

NATIONAL AND INTERNATIONAL MEETINGS

Interim Session, American College of Chest Physicians
Sheraton-Plaza Hotel, Boston, November 27-28, 1955

Fourth International Congress on Diseases of the Chest
Council on International Affairs
American College of Chest Physicians
Cologne, Germany, August 19-23, 1956

POSTGRADUATE COURSES

10th Annual Postgraduate Course on Diseases of the Chest
Hotel Knickerbocker, Chicago, Illinois, October 3-7, 1955

8th Annual Postgraduate Course on Diseases of the Chest
Park-Sheraton Hotel, New York City, November 14-18, 1955

CHAPTER MEETINGS

Pennsylvania Chapter, Pittsburgh, September 21, 1955

Kentucky Chapter, Louisville, September 28, 1955

Preparation of Manuscripts

DISEASES OF THE CHEST, the official journal of the American College of Chest Physicians, publishes manuscripts dealing with tuberculosis, non-tuberculous diseases of the chest and cardiovascular diseases. Kindly send all manuscripts to:

JAY ARTHUR MYERS, M.D., Editor-in-Chief
1316 Mayo Memorial Building
University of Minnesota
Minneapolis 14, Minnesota

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1) All manuscripts should be typewritten on white paper, 8 to 8½ by 11 inches, double or triple spaced. Tables may be single spaced, if necessary. Only one side of the paper should be used. The original copy must be submitted, and the carbon copy should be retained by the author to compare with the proofs. Manuscripts must be original, not published elsewhere, except when special permission is granted by the Editorial Board of *Diseases of the Chest*.

2) The pages should be numbered, preferably at the top right-hand corner. The name of the author should appear on each page of manuscript and on each illustration, chart and table.

3) All dates should be written as follows: *August 25, 1951*—not 8-25-51.

4) Abbreviations should not be used in the manuscript, such as R. U. L., which should be written as *right upper lobe*.

5) Illustrations should be unmounted and appropriately numbered in pencil on the back. Legends should be listed on a separate sheet at the end of the manuscript. Photographs should be black and white glossy prints, not smaller than 3 x 3 nor larger than 5 x 7 inches. Charts and graphs should be drawn on white paper with black India ink. Whenever possible, they should be made by professional medical illustrators.

6) Written permission must accompany identifiable photographs of patients.

7) Four illustrations may be published with each article without charge. Additional photographs, when approved by the Editorial Board, may be published upon payment by the authors or the institution where the work was done.

8) Usually, long lists of references are not necessary or desirable. For most manuscripts, 10 well selected references are adequate.

9) Every paper should contain a summary in English which will be translated at the office of the Managing Editor into Spanish and French. Summaries should be brief, and contain the salient points presented in the paper in 1, 2, 3 order.

10) Authors will be given an opportunity to order reprints when they receive galley proofs for final corrections.

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- *PARASAL SODIUM tablets,* 0.69 Gm. (sod. P.A.S.).

References

(1) V.A. Quarterly Prog. Report, April '53

(2) V.A. Quarterly Prog. Report, Oct. '54

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